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THE DISEASES OF INFANTS AND CHILDREN

By

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WITH 436 ILLUSTRATIONS
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DISEASES OF THE RESPIRATORY SYSTEM

These are among the commonest of the disorders of early life, being exceeded in infancy only by digestive disturbances, and in early childhood even surpassing these in number. Some forms of disease belonging in part here have already been described elsewhere, such as adenoid growths of the nasopharynx and membranous laryngitis dependent upon diphtheia.

CHAPTER I

COUGH

Although but a respiratory symptom cough is one of such importance and produced by such diverse conditions that a brief review of its causes and indications is of importance, by way of introduction to diseases of the respiratory apparatus. It has already been referred to briefly in discussing Significance of Symptoms (Vol. I, p. 23).

Cough is an expulsive effort of respiration of such force and suddenness that the characteristic sound is produced. The effort is set going by a pathological stimulus of the filaments of the pneumogastric nerve terminating in the respiratory mucous membrane involved, or reflexly through the irritation of branches of this or of other cranial nerves supplying other affected regions. Along these afferent filaments the sensation is conducted to the medulla and the expulsive effort set going from this centre through the action of the muscles concerned in respiration. In some cases this appears to depend upon irritation of the nerve-centres solely, without peripheral disturbance. This is seen, for instance, in cough of a hysterical nature or occurring as a habit merely. The distribution of the pneumogastric and communicating nerves being so wide and diverse, the causes of cough may be extremely varied. In some instances it results from direct stimulation by secretion resting upon the respiratory mucous membrane in some part of its course or in the pharynx, or from the presence of a foreign body in the respiratory tract. It is then a real effort to expel a removable offending substance, and is of a serviceable nature and may need to be encouraged. With these exceptions it is for the most part necessarily unproductive and useless.

The following conditions may be mentioned in which cough occurs:

In *bronchitis* it is at first frequent, severe and unproductive. Later it is loose and sometimes only relieved when the secretion is expelled into the pharynx and either expectorated or swallowed. It may be severe enough to cause vomiting, and often produces soreness in the muscles of the chest and abdomen. *Tracheitis*, when occurring alone, produces a cough of a similar nature. In *catarrhal laryngitis* it is at first dry, and later loose as in bronchitis. The hoarseness which accompanies it serves to distinguish it. *Spasmodic laryngitis*, on the other hand, occasions a cough of a peculiar character, loud, barking and ringing. This is not paroxysmal, but comes on especially at night with the symptoms of croup. *Diphtheritic laryngitis* gives rise to a cough very like that of catarrhal laryngitis although with more of a croupy character. In *pneumonia* and *pleurisy* the cough is usually very short and often suppressed and evidently painful; sometimes frequent and harassing. Pleural effusion may produce a very persistent and troublesome, short, dry cough without the element of pain.

Pharyngeal irritation is one of the most common causes, giving rise to a cough which is very frequent and hacking, especially at night, and with a constant sensation of tickling in the throat. It may be due to the reflex irritation arising from adenoid growths of the nasopharynx or of the base of the tongue, or arises in the pharynx itself through the accumulation of secretion on the posterior pharyngeal wall or be brought about by the tickling sensation caused by an elongated uvula. It is often troublesome and resistant to treatment. An *ear-cough* sometimes arises from the presence of foreign bodies or of wax in the auditory canal or the existence of an external otitis.

Cough of a peculiarly paroxysmal character is heard in *pertussis*, but may sometimes occur in severe tracheo-bronchitis and is then confusing from a diagnostic point of view. A somewhat similar cough occasionally accompanies pressure by *enlarged bronchial glands*, or by an *abscess* produced by spinal caries. The condition often described as *spasmodic nocturnal cough*, or *periodic night-cough*, heard frequently in older children, may be due to enlarged bronchial glands or abscess, or to adenoid growths or an unrecognized rhinitis. There is also in many cases a strong nervous element combined with the causes mentioned, or acting alone. This cough occurs in paroxysms, chiefly or solely about the middle of the night or soon after going to bed; or the attacks may repeat themselves several times during the night and occasion much loss of rest. The disease may extend over months.

"*Stomach-cough*" is a term indiscriminately used and too widely applied. There are, however, frequent cases in which cough accompanies indigestion and disappears when this ceases. In some this is probably dependent upon the complicating pharyngeal irritation so often present. In others it appears to be a direct reflex from the stomach. This latter is heard, for instance, in some infants who suffer from cough when the stomach is over-distended from nursing. A short stomach-cough may also be observed at times just before an attack of vomiting occurs.

Cardiac cough is produced chiefly by passive congestion of the lungs, the result of valvular disease of the heart. It may also be heard occasionally in pericarditis. It is hard and usually dry in character.

Finally there is to be considered the *nervous* or *hysterical cough* encountered generally in later childhood especially as puberty is approaching. It is dry, noisy, harsh, forced and frequent, and usually not paroxysmal; does not occur during sleep; and is increased by excitement. It may disappear for months if the general nervous state of the child improves, but reappears readily with the return of nervous disturbance, or with the development of anemia.

In this connection may be mentioned the infrequency with which sputum is expectorated in infancy and early childhood (see Vol. I, p. 560) and the comparative rarity of **hemoptysis** in early life. This is seen occasionally in pneumonia and tuberculosis, oftener in pertussis, and may be severe in gangrene of the lung. Fatal hemoptysis is exceedingly uncommon. Magruder[1] refers to 33 cases collected by Meusnier[2] and adds 5 others including 1 of his own. The condition has since been studied by Rolleston and Robertson-Ross.[3] I have seen an instance of fatal hemoptysis in an infant of 14 months. (See Vol. I, p. 552.)

Treatment of Cough.—The treatment of these different varieties of cough is that of the original cause. Only to be said here is that when the symptom is dependent upon acute inflammation of the larynx, trachea or bronchi sedatives are often of value, but usually after the first few days those remedies are indicated which favor secretion. In cases of other nature expectorants are of no benefit. Nasopharyngeal and pharyngeal irritation is always to be sought for, and local treatment of these conditions, including remedies to diminish the size of an elongated uvula, will often promptly relieve cough over which sedatives have practically no control. Inhalations of water-vapor from a quart (946) of hot water to which dr. 1 (3.9) or less of menthol has been added often gives relief. In the periodical nocturnal cough and in the nervous cough the general health is to be cared for, the pharynx kept from becoming dry at night-time, and such sedatives as bromides administered. In some cases of very persistent cough quinine has proved of benefit.

CHAPTER II

DISEASES OF THE NOSE

MALFORMATIONS

Deviations of the septum nasi are usually the result of injury or of faulty development of the adjacent structures. In the first class there may be an accompanying alteration in the shape of the nose as seen from without. **Depression of the bridge** of the nose may be the result of injury or of the destructive action of hereditary syphilis, or may be of a congenital nature.

Stenosis of the nasal passages or even entire **closure** of these may be found of congenital origin; manifesting itself, if bilateral, by inability on the part of the infant to suck, or, if partial, by evident difficulty in sucking or by evidences of increasing inanition. Examination with the sound will reveal the obstruction, which may be membranaceous or oftener bony in nature. I have seen 1 instance of infantilism associated with complete bony obstruction of the nares. Among other deformities to be mentioned are the broadening of the bridge and the upward tilt of the nostrils seen in *cretinism;* and the narrow nasal fossæ, small nostrils and pinched appearance of the nose often present in *adenoid growths.*

The treatment of nasal deformities is chiefly operative and is described in special treatises on Diseases of the Nose. It is important that operation be done early in all severe cases lest malformations of the chest, the palate, the jaws, and even of the skull develop.

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[1] Arch. of Ped., 1908, XXV, 518.

[2] Thèse de Paris, 1892.

[3] Brit. Jour. Child. Dis., 1914, XI, 407

ACUTE RHINITIS

(Coryza)

Etiology.—The tendency to develop "cold in the head," one of the most common affections seen in children, begins promptly after birth and continues active throughout the rest of life. In certain individuals, and even in certain families, there is a special predisposition to it; and the influence of inheritance is sometimes very noticeable. The exudative diathesis is a predisposing cause. Many diverse influences, however, are the more active agents. Local chilling is undoubtedly one of these, and frequently this needs to be but slight and of very brief duration to produce a severe cold. Should the chilling immediately follow overheating of the body the susceptibility is then much greater. For this reason dressing too warmly is a common cause, on account of the excessive perspiration which is produced by any slight exercise and the subsequent chilling which comes with rest. Insufficient protection by clothing is equally harmful, but less frequently operative. Draughts upon the head; the absence of sufficient covering for the arms and legs subjecting them to sudden chilling; getting the feet wet; the exposure of the abdomen through the absence of a binder in infants in short clothes; the playing upon the floor in winter time, and many similar conditions are important factors. The influence of season is decided; autumn and spring especially predisposing since sudden changes of temperature are most likely to occur at these times. The damp, raw weather accompanying a thaw in winter, or days with high cold winds are a determining cause. Any debilitating or sensitizing influence may occasion constantly recurring attacks. Here may be mentioned close confinement to the house with insufficient air and ventilation in the living and sleeping rooms, the custom of giving the bath of too high a temperature with consequent relaxation of the skin, the presence of adenoids or of other conditions chronically obstructing nasal respiration, and the existence of rickets or other debilitating disease. Rhinitis is a constant attendant upon certain diseases such as nasal diphtheria, grippe, and especially measles. Certain drugs may produce coryza, especially to be noted being the iodides administered internally; while local irritants, as the pollen of certain plants, the odor of roses, the volatile emanations from horses or some other substance to which the patient shows an idiosyncrasy, may produce an attack. (See also Hay Fever, pp. 7 and 42.)

That coryza often attacks several members of a family in quick succession suggests the very probable influence of contagion, which is without doubt operative in many instances; although the fact that all may have been exposed simultaneously to some other cause, such as changes in the weather, is constantly to be borne in mind in drawing conclusions. Undoubtedly the action of bacteria of various sorts is an important element, but the coöperation of other factors is usually required. There is probably first produced a congestion of the mucous membrane dependent upon some other cause, and this permits of the action of the bacteria. Various microörganisms have been found associated with rhinitis, among them the streptococcus, staphylococcus, pneumococcus, micrococcus catarrhalis and the bacillus rhinitis (Tunicliffe).[1] The specific forms which produce measles, grippe and diphtheria, appear to be sufficient, acting alone, to produce rhinitis, and this condition is often the first symptom of these diseases.

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[1] Journ. Infect. Dis., 1913, XIII, 283; 1915, XVI, 493.

Symptoms and Course.—Following the increased redness and swelling of the mucous membrane there occurs an augmented secretion from the nose of fluid of the normal character. In the milder cases this may subside in a few hours and the attack be over. In other more severe ones the congestion grows greater, there is frequent sneezing, the discharge is irritating and reddens the edges of the nostrils and the upper lip, respiration through the nose becomes almost or quite impossible, and the general condition is one of extreme discomfort. The compulsory breathing through the mouth produces a sensation of sore throat through the drying which results; and in the case of infants mechanically interferes with the taking of food, since the child cannot suck and breathe through the mouth at the same time. The temperature is seldom more than slightly elevated except in young infants. After a day or two the discharge becomes more seropurulent and finally of a green mucopurulent character often more or less tinged with blood. Attending the rhinitis or sometimes preceding it there is in the severer cases involvement of the lachrymal duct and conjunctiva, with consequent lachrymation and slight conjunctivitis. The inflammation may spread through the Eustachian tube to the ear, and otitis, often purulent, develop. The accessory sinuses are not as often involved in childhood as in later life. Kelly[1] could collect but 18 cases of empyema of the antrum occurring in infants; and Onodi[2] could find in medical literature only 53 instances of disease of any of the accessory sinuses developing before the age of 10 years. Phelps[3] and Skillern[4] both emphasize the infrequency of sinusitis in infancy and early childhood, owing to the imperfect development of the sinuses at this period. Not infrequently the inflammation attacks simultaneously or in sequence the nose, pharynx, larynx, trachea and bronchi; in many cases pharyngitis is the first manifestation, and rhinitis develops subsequent to this.

Course and Prognosis.—In the milder cases the congestion is but temporary and disappears nearly or completely after a few hours or a day. In others a moderate congestion lasts a couple of days, but the discharge does not reach a purulent state. In the fully developed attack it may be a week or more before the condition returns to normal. When the necessary underlying requirements are present the coryza may assume a subacute or chronc form. The danger of the development of otitis is very great, especially in infancy and early childhood. In infancy the nutrition may be injured by the interference with nursing, and death may result from this cause in infants previously debilitated. The tendency for a bronchopneumonia to follow coryza is also to be borne in mind.

Diagnosis.—Simple coryza is to be distinguished from that symptomatic of other affections, such as measles, grippe and diphtheria, by a careful search for the special symptoms of these diseases. The persistence of high temperature renders one always suspicious that the coryza is not a primary disorder, and the possibility of grippe or measles will be entertained. The discharge of nasal diphtheria is early bloody and very irritating to the lips, is unattended by the complicating conjunctivitis or the other symptoms of simple acute rhinitis, and is of longer continuance. Examination of the nose may reveal a pseudomembrane, but in suspicious cases cultures should be taken promptly.

[1] Edinb. Med. Jour., 1904, LVIII, 302.
[2] Jahrb. f. Kinderh., 1915, LXXXI, 159.
[3] Arch. of Ped., 1917, XXXIV, 520.
[4] Journ. Amer. Med. Assoc., 1917, LXIX, 895.

Treatment. **Prophylaxis.**—Particular attention should be given to this. The possibility of one member of a family acquiring the disease by association with an infected member is to be borne in mind, and such contact avoided, particularly in the case of infants. Predisposing causes of any nature must be diligently sought for. The hygienic conditions must be improved on the lines indicated in discussing etiology. This is particularly true where repeated attacks of coryza are frequent. It is only by careful attention to the details of exercise, pure air in the sleeping room, proper clothing, house temperature, and the like, that avoidance of coryza can be expected. Of especial moment is life largely in the open air. The hours chosen for bathing, giving food, and the morning nap are often such that in winter time an infant, as a result, spends all or the greater part of the morning indoors; while the daylight of the afternoon is so short and the weather so uncertain that the outing is often missed entirely or is very greatly curtailed. Some rearrangement of the life is necessary in such cases. Moreover, it is not essential that the day be perfectly clear to permit the taking of a young infant out of doors; nor is it imperative that the morning nap be in the house. High, cold winds are, however, to be avoided. In country houses the sheltered side of a veranda offers a suitable place under such conditions, while in cities the young infant may be dressed in its outdoor clothing and put in its coach in a room with the windows open wide. Older children should be accustomed to go out properly protected in almost any sort of weather. The importance of suitable clothing, too, to which reference has been made under etiology, is very great. More children are dressed too warmly than the reverse. It is better to have the arms and legs always covered except in the hottest weather, but the clothes should be of only moderate weight. Then, when there occurs a sudden change in the weather, or when a room appears unduly cool, an outside wrap of some sort may be worn. To dress a child with underclothing warm enough for all occasions means that too often it will be overheated and freely perspiring.

Naturally every means must be taken to improve the general health if this is below normal. A cool bath given before breakfast and followed by brisk friction will be found an excellent measure to harden a child, but efforts at hardening by improper exposure do harm rather than good. Tonic treatment may be required. Finally, adenoids present must be removed if coryza is frequent.

Treatment of the Attack.—When seen early, the patient should be given thorough purgation, a warm general bath or hot mustard foot-bath, and be kept in bed in the effort to produce a sweat. Later the child should at least be kept quiet and in an evenly heated, rather warm room, and in the winter off of the floor. The air, however, must be fresh. The diet should be restricted, particularly if there is an elevation of temperature. For local use, a spray of menthol and liquid petrolatum (gr. 2 or 3 : oz. 1) (0.13 or 0.19 : 30), or for infants without menthol, often gives relief; or, if the children object to the use of a spray, a few drops may be instilled from a dropper, the head being bent far back at the time in order that the fluid shall not run along the floor of the nose to the throat and thus fail to reach the affected parts. The instillation of 2 or 3 drops of a 2 per cent. solution of cocaine in each nostril will, it is true, relieve the congestion in a surprising manner and give temporary relief to the obstruction to respiration. The remedy is, however, a dangerous one in infancy and early childhood and is not to be recommended for frequent use even after this period. The temporary relief is of short duration and is followed by increased swelling. A safer application to accomplish the result is of epinephrine of a strength not less than 1 : 3000. The inhalation of vapor from hot water on which a few crystals of menthol have been placed is also serviceable. Cold cream or a boric acid ointment should be applied to the upper lip and the edges of the nostrils. Internally atropine is of value in checking the discharge and relieving the congestion, especially in the earlier stage, small doses being given frequently according to the effect produced. In some individuals, quinine seems to have a special power in curtailing or aborting an attack, but this is oftener the exception than the rule. Hexamethylenamine has also been found of service in the hands of many. In severe cases, or in those in the later stages, a spray or gentle douch of warm liq. sod. boratis comp. (Dobell's solution), or of warm normal saline solution, is useful for cleansing purposes, followed by the instillation of a few drops of a solution of argyrol in 10 per cent. to 25 per cent. strength. The treatment may be given 3 or 4 times a day.

Hay Fever.—This title designates a form of acute rhinitis recurring at the same season of each year, the season varying with the individual. It is brought about by the action of various local irritants, such as the pollen of different plants, the odor of roses, violets, peaches, etc., and the emanations from certain animals, as the cat or dog, and especially the horse. It would seem probable that all of these effects are instances of an anaphylactic reaction to certain proteins. The number of plants which may produce this vernal or autumnal catarrh is large. This phase of the subject has been studied with especial care by Dunbar[1] who believes that the active cause in most cases is a toxalbumin contained in the pollen grains. Abnormalities of the mucous membrane of the nose may be a predisposing cause. An individual susceptibility is necessary and the patients are generally of a highly nervous temperament. Inheritance is frequently observed. The disease is not common in early life, and this is especially true of the first 3 or 4 years.

The **symptoms** come on suddenly after exposure, and increase in severity if this continues. They are those of severe coryza, accompanied by conjunctivitis. There is an intense itching in the nose and eyes; profuse watery nasal discharge; frequent violent sneezing; lachrymation, and photophobia. As the patient grows older and the annual attacks have been repeated, asthma is liable to be associated with the rhinitis, and sometimes even largely to replace it.

The **prognosis** for ultimate recovery is poor. The **treatment** is largely prophylactic. When possible the child should be removed during the season of danger to a region which experience shows renders him free from the disease. This should be done before the season for the attack arrives. For the direct treatment of the disorder little can be done except the employment for palliative purposes of the measures recommended for ordinary severe acute rhinitis. Cocaine should never be employed. The temporary relief given by it is followed by increasing congestion; and there is great danger of establishing a habit. The value of immunizing is still uncertain. It has been successful in some cases; useless in others.

Membranous Rhinitis.—The disorder of this nature dependent upon diphtheria has already been discussed. (See Diphtheria, Vol. I, p. 449.) There is rarely seen a similar condition due to the streptococcus, staphylococcus, pneumococcus, or pseudo-diphtheria bacillus. The local symptoms are similar to those of diphtheritic rhinitis; and even the failure to discover the diphtheria-bacillus does not make it certain that this is not the condition present.

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[1] Modern Medicine, Osler and McCrae, 1914, II, 855.

CHRONIC RHINITIS

Chronic rhinitis may assume one of several forms, and may be consequently divided into: (1) Simple Chronic Rhinitis; (2) Hypertrophic Rhinitis; (3) Atrophic Rhinitis; (4) Syphilitic Rhinitis.

1. SIMPLE PURULENT CHRONIC RHINITIS

(Chronic Nasopharyngitis; Post-nasal Catarrh; Chronic Nasal Catarrh)

Etiology.—This common disorder in early life may follow repeated attacks of acute coryza, these depending usually upon the presence of adenoids, or less often of polypi; or be the result of the introduction of foreign bodies into the nose, deviations of the septum, and the presence of the exudative-lymphatic diathesis. (See Exudative Diathesis, Vol. I, p. 630; Tuberculous Adenitis, Vol. I, p. 557.) Often the children appear to be in perfect health in other respects.

Pathological Anatomy.—There is no such engorgement of the erectile tissue of the nose as occurs in the acute cases, and consequently no such interference with respiration. The pathological condition is rather a profuse mucopurulent secretion.

Symptoms.—There is a more or less constant discharge from the nose of a secretion which is mucous, or oftener mucopurulent or purulent. Children old enough to blow the nose remove the secretion readily, but it soon reaccumulates. There is redness and excoriation of the upper lip and the edges of the nostrils. These parts may be decidedly swollen in the severer cases, and eczema of the upper lip may develop. The injection of the eyes and other symptoms characteristic of acute rhinitis are absent, but there is a frequent association of redness of the eyelids and of eczema, dependent upon the exudative diathesis as a common cause.

Course and Prognosis.—Cases dependent upon the presence of foreign bodies are usually cured promptly by the removal of these. When from other causes the disease may continue indefinitely, lasting a month or extending over years; perhaps being better in summer and returning in force in winter. If a purulent secretion is long-continued the mucous glands may gradually atrophy, the secretion dries and forms crusts upon the mucous membrane, and the atrophic form of chronic rhinitis develops. Otitis, pharyngitis, or enlargement of the glands of the neck may occur as complications.

Diagnosis.—The recognition of the cause is the first indication. The presence of a foreign body produces a purulent and sometimes blood-stained discharge upon one side only, and examination with a probe will reveal the obstruction. Hereditary syphilis exhibits other characteristic evidences; while the complex of symptoms present makes readily recognizable the cases dependent upon adenoids, or those often associated with tuberculous adenitis. The distinction from hypertrophic rhinitis is to be made by the greater degree of stenosis and the lesser of secretion present in the latter disease, but the differentiation is often not easy.

Treatment.—Preventive treatment is of the utmost importance. The prophylaxis already outlined under Acute Rhinitis is to be carried out carefully to prevent the recurrence of the acute attacks to which the chronic form may be a sequel. The administration of cod-liver oil, iron, arsenic or the iodides is indicated if there is debility with anemia. In the treatment of the condition already established, the search for a foreign body should be made, and this removed. The removal of adenoid growths or of polypi present is also essential. Apart from the treatment of any direct cause, the nasal passages must be kept as free as possible of secretion, by the employment twice daily or oftener of douching or syringing with some warm alkaline antiseptic solution, such as the liq. sod. borat. comp. (Dobell's solution). There is some danger of this driving the secretion into the Eustachian tube, but care in the treatment, with the employment of but very little force, will avoid this. The nasal douche applied by gravity should be under very low pressure, the vessel being elevated hardly at all above the level of the nose. While it is being given, the patient should extend the head slightly forward and breath through the opened mouth. In place of this the fluid may be applied by an atomizer 3 or 4 times a day, spraying and very gentle blowing of the nose alternating several times during each treatment. This cleansing may be followed by a spray of some gentle astringent such as diluted fluidextract of hamamelis, or of a solution of sulpho-carbolate of zinc (gr. 2 to 5 : oz. 1) (0.13 to 0.32 : 30). Finally one of menthol in liquid petrolatum may be employed. Sometimes good follows the application within the nostril of an ointment of calomel gr. 1 (0.07) and petrolatum dr. 1 (3.9).

Constitutional treatment is often even more important than local measures. It is frequently only after the general health has been improved by appropriate remedies that relief of the local condition can be expected.

2. HYPERTROPHIC RHINITIS

Etiology.—This disease is not so common in children as is the simple chronic rhinitis, and in early childhood is almost unknown except perhaps in a mild form. It is usually preceded by numerous acute attacks of rhinitis, which may be the cause, or perhaps oftener are only a symptom, of the hypertrophic condition. It is commoner in cold, damp climates, and is the result, too, of the irritation from continued inhalation of dust; but the most frequent cause is chronic nasal obstruction of some sort, as from adenoids, or particularly from deviations of the septum. It develops especially, too, if there is associated impairment of the general health.

Pathological Anatomy.—In well-developed cases there is hyperplasia of the nasal mucous membrane, particularly of that covering the turbinated bones, to such an extent that the space for breathing is greatly encroached upon. Both mucous and adenoid tissues are hypertrophied, the submucous tissue proliferated and infiltrated, and the blood-vessels constantly engorged. The changes have become so permanent that discharge of the secretion can occur only to a moderate extent at any time. There is little if any involvement of the accessory sinuses, owing to the imperfect development of these before puberty. In the milder cases, which are those oftenest seen in childhood, the engorgement of the vessels predominates over infiltration and proliferation. Consequently improvement at intervals in the symptoms is possible.

Symptoms.—The chief manifestation of the disease in childhood is persistent, more or less complete obstruction to respiration on one or both sides, with which is associated nasal discharge which is mucopurulent but usually less abundant than in simple chronic rhinitis. The secretion is often chiefly post-nasal and drops into the throat. The degree of stenosis varies from time to time, sometimes one side being affected, and for a short time, the other being meanwhile more or less free. Thus while lying upon one side in bed, the lower nostril becomes occluded, to open again when the patient turns to the other side. Obstruction may grow worse also under such conditions as exposure to the air of hot, dry rooms, and the like. Many secondary symptoms of stenosis arise, such as mouth-breathing, with constant irritation of the throat and pharyngeal cough; attacks of asthma or of croup; bronchitis; deafness; alteration of the voice; disturbance of sleep; nervousness; restlessness; headache; and mental backwardness. These secondary symptoms are in fact very similar in number and variety to those resulting from the presence of adenoids, which lesion, with enlargement of the faucial tonsils, is often combined with hypertrophic rhinitis. Examination of the nares reveals the thickened, swollen condition of the covering of the turbinated bones.

Course and Prognosis.—There is no natural limitation to the duration of the disease, which tends to get worse rather than better unless the cause is successfully removed. With this removal and under careful continued treatment the majority of moderate cases will recover; but the severer ones will need radical therapeutic measures.

Diagnosis.—The distinction is to be made especially from the presence of adenoid vegetations. Rhinoscopic examination in hypertrophic rhinitis will show the covering of the turbinated bones greatly thickened, pink in color, obstructing the nasal passages, and very resistant to pressure by a probe. Simple chronic rhinitis has usually a freer discharge with no evidence of hypertrophy on direct examination and less nasal obstruction.

Treatment.—As with other forms of rhinitis prophylaxis is important and consists in the removing of all causes liable to produce recurring attacks of acute coryza (see p. 4), including the restoring or maintaining of the best condition of the general health. For the disease already established, although the sustaining of the general health is necessary, local treatment of the hypertrophic state is required. This is best undertaken by a specialist in diseases of the nose. Hypertrophied adenoid tissue and enlarged faucial tonsils must be removed as a preliminary measure. This of itself may result in the cure of the rhinitis. In addition, measures should be employed to maintain a state of cleanliness and to reduce the hypertrophy. For the former the alkaline antiseptic spraying or syringing followed by oily applications should be employed as recommended for simple chronic rhinitis. In place of menthol, the oleum pini Canadensis m. 5 (0.31) or thymol (gr. 1) (0.07) with petrolatum liquidum fl.oz. 1 (30) may be used. In other cases weak astringent aqueous sprays are of advantage, such as sulphocarbolate of zinc (gr. 5 : oz. 1) (0.32 : 30). Often serviceable is the application of iodine to the diseased region (iodine gr. 5 (0.32); potassium iodide gr. 24 (1.56); glycerin fl.oz. 1 (30). This may be used once or twice a week after thorough cleansing.

In all well-developed cases, however, more radical measures are required, including cauterization of the hypertrophic tissue with the galvanocautery or chromic acid. The treatment can be carried out properly only by one specially trained.

3. ATROPHIC RHINITIS

Ozena

Etiology.—The cause of the condition has been much discussed. In some cases it may be a sequel to simple purulent rhinitis of long duration; in others it succeeds the hypertrophic form of the disease; in still others the atrophy appears to be a primary process. It is rarely seen before later childhood and is more frequent in females. There is active also the powerful influence of the constitutional condition of the subject, such as anemia and especially glandular tuberculosis; and the influence of heredity is sometimes observed.

Pathological Anatomy.—The lesions consist of atrophy of the entire mucous membrane, with alteration of the epithelium from the cylindrical to the squamous type; degeneration of the cells; a decrease of the mucous glands and adenoid tissue, and shrinking of the blood vessels. In advanced cases there is involvement of the cartilaginous and bony structures. As a result of the shrinking of the mucous membrane all the nasal passages and openings are unusually large.

Symptoms.—What little nasal secretion occurs forms large, firmly adherent crusts which are removed imperfectly if at all by blowing the nose or sneezing, owing to the large size of the nasal passages. Under the influence of bacteria, these crusts decompose and generally produce an excessively offensive odor, which is the chief characteristic of the disease. After the decomposition has softened them the crusts are discharged, often leaving superficial ulcerated areas. The patient's own sense of smell is diminished or lost and he is not himself conscious of the odor which surrounds him. There is no obstruction to the nasal respiration except in cases where the crusts have become unusually large and thick. Rhinoscopic examination shows unusually wide passages, with disappearance to a large extent of the inferior turbinated body. This condition permits of observation of a large part of the posterior pharyngeal wall. In advanced cases there is broadening of the nose as viewed from without, and the atrophy within spreads to the pharynx and nasopharynx, with dryness of the mucous membrane and the formation of crusts there. Irritation of the pharynx and larynx are also produced by the constant entrance of air not sufficiently moistened in its passage through the nose.

Course and Prognosis.—The disease lasts an indefinite time with no tendency to spontaneous improvement. Cure of the atrophy already present is hardly possible, but mild cases may have further progress arrested by systematic, long-continued treatment. It is noteworthy that deafness is not a common complication, but some degree of chronic laryngitis is frequent.

Diagnosis.—The unusually wide nasal passages, the presence of firm, hard crusts, and the odor characterize the disease. Syphilitic rhinitis resembles it in the odor and crusting, but is to be distinguished by the extent of the deep ulceration present, the absence or slight degree of atrophy of the mucous membrane, and the presence of other syphilitic symptoms.

Treatment.—Prophylaxis is to be sought in the prompt and thorough treatment of a simple chronic or of a hypertrophic rhinitis, which might be the precursor. In the treatment of the atrophy already present cleanliness of the nasal passages is the first object. The nasal douche or syringe should be used twice a day at least, according to the method spoken of under simple chronic rhinitis (p. 8); employing an antiseptic alkaline fluid such as liq. sod. borat. comp. or liq. antisepticus diluted with 3 or 4 parts of water. The fluid should be warm, and sufficient of it used to soften and remove any crusts present, 1 or 2 pints (473 or 946) being required. There is some risk of producing involvement of the ear, but this is one which must be taken. The atomization of peroxide of hydrogen diluted with 3 parts of water is also serviceable in softening the crusts. If these are firmly attached forcible removal of them with cotton on an applicator will be necessary. This and, in fact, all local treatment of the disease, is best carried out by a specialist for affections of the nose. Following cleansing, spraying should be done with menthol in liquid petrolatum (gr. 3 : fl. oz. 1) (0.19 : 30). A serviceable remedy used on cotton with an applicator is iodine (iodine gr. 8 (0.52); potassium iodide gr. 24 (1.56); glycerin fl. oz. 1 (30)); or aristol powder may be employed by insufflation, followed by an oily spray.

Equally important is constitutional treatment. Anemia and other evidences of impaired health must receive appropriate remedies. Cod-liver oil is often of benefit in this connection. All hygienic measures possible to aid in the improvement of health are to be sought.

4. SYPHILITIC RHINITIS

This variety of chronic rhinitis may show itself either at an early age or as one of the later symptoms of syphilis. It has been considered to some extent in the chapter upon this disease. (See Vol. I, pp. 571, 575.)

Pathological Anatomy.—In the early coryza of the syphilitic new born, the lesions are chiefly those of ordinary chronic rhinitis. In later hereditary syphilis, however, they are of a different nature, consisting in the formation of gummata and the subsequent breaking down of these; producing deep-seated, widespread ulceration of the mucous membrane and the destruction of the bones and cartilages, with resulting flattening of the bridge of the nose as seen from without, perforation of the septum, and involvement of neighboring parts.

Symptoms.—Early syphilitic rhinitis produces the "snuffles" so characteristic of this period of the infection. This appears in the early weeks of life. The nose becomes greatly obstructed, making respiration difficult and even interfering with the taking of food. There is an abundant serous or mucopurulent discharge from the nose, often slightly blood-tinged. The upper lip becomes excoriated and sometimes fissures. In the late form of the disease, apart from the deformities which may result, there is a free discharge of pus, often with a very offensive odor if destruction of the bone is going on. Obstruction to respiration may be produced by the pressure of gummata or by cicatrizing processes. In other cases the nasal passages are left unduly wide and the formation of crusts readily occurs.

Prognosis.—This is good in the early cases, so far as the nasal condition is concerned. The disorder lasts a few weeks or months and readily yields to antisyphilitic treatment, in cases in which this is successful for the relief of the general condition. Some permanent alteration of the mucous membrane or even of the shape of the nose may perhaps be detected in later years. The prognosis of the later cases is much more serious. The destruction which has often already taken place when the patient is first seen cannot be repaired.

Diagnosis.—The early cases are readily recognized by the continuance of the condition for a longer period than would be natural for an acute simple rhinitis, and by the presence of other evidences of inherited syphilis. Rhinitis occurring as a late symptom of the disease can scarcely be confounded with any other affection in early life.

Treatment.—Antisyphilitic remedies constitute the principal treatment needed. (See Syphilis, Vol. I, p. 581.) In addition the usual cleansing methods suitable for simple chronic rhinitis are to be employed.

EPISTAXIS

Etiology.—Nose-bleed is a symptom of such varied significance that it warrants separate consideration. It is rare in the new born and in infancy, but common in childhood, and decreases after puberty. Occurring in the new born it is generally dependent upon syphilis. It is especially frequent in school life, induced perhaps by overheated, dry rooms, faulty position of the body producing congestion, and the like. Coryza, coughing, or such traumata as blows and forcible blowing of or picking at the nose may be exciting causes, while chronic rhinitis, adenoids, or small erosions on the mucous membrane are frequent agents in producing recurrent attacks. Foreign bodies in the nose, nasal polypi, mental excitement, the strain of physical exercise, or sometimes the mere bending over of the body, may be sufficient to occasion it. Various diseases are liable to produce epistaxis, prominent among these being affections of the heart, lungs or kidneys, resulting in passive congestion; and it may occur, too, in severe anemia of any form, leukemia, purpura, scorbutus and hemophilia. Certain infectious disorders may also be attended by it, chief among these being typhoid fever, in which epistaxis is an early symptom. It is frequently produced by the passive congestion attending the paroxysms of pertussis, and may occur also in the course of diphtheria, scarlet fever, measles, grippe, and malarial fever. Toward the development of puberty it is not infrequent in girls and may then be a vicarious function. Previous to this time it is more common in boys.

Pathological Anatomy.—The hemorrhage is dependent upon the rupture of a blood-vessel. Sometimes this is situated in a small ulcer on the septum, and recurrences of the bleeding are then due to the giving way of the thrombus which forms after the initial rupture of a small artery. In other cases there is a varicose condition of the blood-vessels. Each of these lesions is usually situated upon the cartilaginous septum well forward, and easily discovered by rhinoscopic examination. Sometimes the nose-bleed takes its origin in adenoid growths in the nasopharnyx.

Symptoms.—There may be a sensation of pain or fullness in the head or of dizziness before the attack, but very often there is no warning whatever. The blood comes in drops from one nostril, generally flows slowly, and the amount is much smaller than the condition of the handkerchief used leads the family to believe. Exceptionally it may be in large amount, as in cases of hemophilia, leukemia, pernicious anemia, or the hemorrhagic form of infectious diseases; or small hemorrhages may be frequently repeated over so considerable a time that decided anemia is produced. In many instances the hemorrhage is concealed and the blood swallowed. This is true, for instance, when the bleeding takes place at night, and it may then be recognized only if blood is vomited or passed by the bowel.

Course and Prognosis.—The hemorrhage usually stops of itself in 10 or 20 minutes or less, but may recur daily or every few days or weeks. It is usually not serious unless the frequent repetition leads to anemia. In some conditions, as in pernicious anemia, leukemia, hemophilia and the infectious fevers of hemorrhagic type, the bleeding may be severe and even fatal. I have seen a fatal issue from expistaxis occur in the course of typhoid fever in a child of 8 years.

Diagnosis.—This is easy except in the cases where the patient lies on the back and the hemorrhage occurs during sleep. Here epistaxis must be distinguished from gastric or intestinal hemorrhage. Examination of the nares will perhaps show the site of the bleeding if it is nasal in origin, or clots of blood in the pharynx may indicate that it arises from adenoid growths.

Treatment.—The first indication is to *stop hemorrhage in progress.* Firm pressure against the jaw in the region just below the nose will often control the bleeding. Ice inserted into the nostril or applied to the bridge is of service, or placed on the nape of the neck may aid through the reflex contraction of the nasal vessels. Compressing the alæ between the thumb and finger is also useful. The child should keep as still as possible, remaining erect in a sitting position with the hands above or behind the head, have the clothing around the neck loosened, and refrain from blowing the nose. This last especially should be avoided, too, after the flow of blood has ceased, lest clots be displaced and the hemorrhage recur. An excellent remedy is the introduction on cotton of a few drops of a solution of epinephrine (1 : 1000), perhaps with 2 per cent. of cocaine added. Should none of these measures avail, it may be possible to detect the bleeding area and to touch it with a caustic of some sort. Plugging the nares must be resorted to if this fails, but is seldom required. Internal medication may be tried in severe cases. For this purpose ergot, calcium lactate, or oil of erigeron, may be used. In obstinate cases the hypodermic injection of horse-serum is often efficacious.

In cases with a disposition to frequent epistaxis *preventive measures* are indicated. Examination should be made for ulceration or varicose condition of the septum and cauterization should be employed, as with nitrate of silver, chromic acid, or the galvanocautery. Constitutional conditions need treatment, hygienic and other measures being used to improve the general health and remove anemia. The clothing about the neck should be worn loose and the position of the child in sitting should be one which does not favor congestion of the nasal mucous membrane. It may be necessary to avoid the hot, dry air of the schoolroom, and to pass more time out of doors. Violent blowing of the nose must be forbidden.

ADENOID HYPERTROPHY

Adenoid growths, being situated in the nasopharynx rather than the nose itself, and being so closely allied to enlargement of the faucial tonsils, have already been described under Diseases of the Pharynx.

MORBID GROWTHS OF THE NOSE

Nasal Polypi.—These growths are not common in children of any age, and are especially rare in infancy and early childhood. They do not develop upon healthy mucous membrane, the most powerful predisposing factor being hypertrophic rhinitis or other cause which produces persistent nasal stenosis with retention of secretion. The polypi are sessile or pedunculated; multiple; of color varying from grey to red, and either myxomatous or oftener fibro-myxomatous in nature, the amount of connective tissue varying with the case. They are situated especially upon the middle turbinated bone, and generally in both nostrils. They vary in size at different times, being larger in damp weather or when the patient has an attack of acute rhinitis.

The **symptoms** consist of stenosis or complete obstruction of the nasal passages; mucous or mucopurulent discharge of moderate amount; and mouth-breathing and its natural results. There are a number of secondary symptoms such as occur in any grave obstruction to respiration. Among these are headache; dullness of hearing, taste and smell; reflex cough; sneezing and asthmatic attacks. The course is longer than in the obstruction due to foreign bodies, and the nasal discharge usually less purulent. Rhinoscopic examination with a probe and mirror will reveal the presence of the growths. **Treatment** consists in removal by the wire snare, forceps, or cautery; combined with attention to the chronic rhinitis or other obstruction which is the predisposing cause. Unless this latter is accomplished satisfactorily the growths may rapidly return.

Other Nasal Growths.—These are all rare in early life. Among those of a benign nature may be mentioned fibroma, papilloma, glioma, and chondroma. Fibromata are those oftenest observed. They occupy the nasopharynx and, if not removed, may grow to large size. Papillomata are generally quite small and seen in the anterior portion of the nares. Gliomata and chondromata are exceedingly uncommon. Of malignant growths sarcoma is the form oftenest encountered, although unusual.

FOREIGN BODIES IN THE NOSE

The introduction of foreign bodies of various sorts into the nose is of common occurrence in early childhood. Among those thus introduced are especially such smooth substances as peas, beans, shoe-buttons, cherry-stones, beads, and the like. The object is situated at first well forward, but unskillful efforts of the patient or others to remove it often result only in pushing it further in. Frequently the child says nothing of the occurrence, either through fear or forgetfulness. The early symptoms are only obstruction to respiration, pain, and perhaps sneezing from the local irritation. If the object is smooth and hard and not of a nature to swell by absorption of liquid it may sometimes remain weeks or months without producing other symptoms. Generally, however, evidences of increasing irritation soon develop, with free purulent nasal discharge, swelling of the mucous membrane, and increased obstruction, such as occurs in simple chronic rhinitis. Ulceration may later take place, and the discharge become bloody and offensive. The fact that the affection is always unilateral strongly suggests the presence of a foreign body, and examination with a probe and speculum will generally reveal it readily. Removal is usually easy. Forcible blowing of the nose with the healthy nostril compressed is sometimes effective. If the body is firmly fixed, cocaine may be applied and forceps employed. After-treatment is required for a few days at most, and is that indicated for chronic rhinitis.

CHAPTER III

DISEASES OF THE LARYNX

ACUTE LARYNGITIS

Several forms may be classed together under this heading, inasmuch as they shade into each other to a certain extent, the different elements which are more or less combined to produce the symptoms varying with the individual case. As a result the number of titles employed to designate the different types is large and confusing, and any division is to an extent arbitrary and to be used only as an aid to study. With this, then, in view the disease may be divided into: (1) Catarrhal Laryngitis; (2) Spasmodic Laryngitis; (3) Subglottic Laryngitis.

1. ACUTE CATARRHAL LARYNGITIS

Etiology.—The disease may occur at any time in early life, but is less common in children than in adults. It is seen perhaps oftener in later childhood than before this period. The attack may be secondary to various other diseases such as rhinitis, pharyngitis, scarlet fever, grippe and especially measles, or be an exacerbation of a more chronic condition; or it may be primary, brought on by exposure to chilling or other defective hygienic conditions, as in acute rhinitis; or by the irritation of the larynx by excessive crying or shouting, or the inhaling of irritating substances, such as coal-gas, dust, steam, and the like.

Pathological Anatomy.—The lesions are entirely catarrhal in nature. The mucous membrane of the epiglottis and the arytenoid cartilages is swollen, hyperemic, and at first dry but later secreting tenacious mucus or muco-pus. Any or all regions of the larynx may be involved. The vocal cords may be almost normal, or may be injected or less frequently swollen; but oftenest the laryngeal inflammation is limited to the region above the vocal cords.

Symptoms.—This form of the disease is the mildest variety of laryngitis. The chief symptoms are hoarseness of the voice and a dry, hoarse and, later, loose cough, without the peculiar barking character of croup. In the lighter cases the general condition is usually unaffected, the appetite is good, and there is little or no fever. In the severer ones the temperature is 101°F. (38.3°C.) or over, and the child may feel ill. The hoarseness may vary in degree up to complete aphonia in the severe cases, while in the milder ones it may be scarcely noticeable. There is no dyspnea. Rhinitis nearly always occurs in conjunction with or antedates the laryngitis and involvement of the trachea to some extent is frequent.

Course and Prognosis.—The duration is usually short, equalling from 3 to 8 days, the cough growing gradually looser and the hoarseness lessening. The prognosis of the disease itself is entirely good, but such complications as pneumonia or bronchitis may develop, or the disorder may be a precursor of the more severe form of laryngitis to be described (subglottic laryngitis).

Diagnosis.—This is entirely easy in uncomplicated cases, only rendered difficult by the tendency for the disease to be combined with the element of spasm seen in spasmodic croup, or to pass into the severer form of laryngitis where great swelling and dyspnea occur.

Treatment.—The same general methods of treatment are indicated as in acute rhinitis. The most important is rest of the larynx, and talking should be avoided. The child should be kept in a room well-ventilated but rather warmer than usual, preferably with the air moistened with water-vapor. (See Vol. I, p. 235.) If there is any fever, a purgative and a hot bath should be given and the patient kept in bed. The continued application of warm or cold compresses over the larynx is useful in many cases. In other instances external irritation over the larynx may be employed, using weak mustard-plasters, camphorated oil, soap-liniment, etc. After the action of the laxative, opium, aconite and ipecacuanha may be administered internally in divided doses appropriate to the age.

2. SPASMODIC LARYNGITIS

(False Croup; Spasmodic Croup; Stridulous Laryngitis)

In this disease is especially active the element of spasm of the muscles of the larynx. All degrees of this may exist, there being sometimes great spasm and little or no catarrhal inflammation, or in other cases chiefly the latter with only slight tendency to symptoms of croup. Thus catarrhal and spasmodic laryngitis shade into each other by many gradations.

Etiology.—The same causes operate as in simple catarrhal laryngitis. In addition are seen certain other etiological factors, especially the influence of age. Croup is uncommon in the first 2 years of life, but frequent between the ages of 2 and 4 years. There is a decided individual predisposition, some children never suffering from it no matter how severe a catarrhal laryngitis may develop; others showing a great proneness to recurrences on very slight cause. It is especially in these latter that the development of attacks of croup is liable to be witnessed even through later childhood. The same predisposition may affect families, a number of the children being prone to suffer attacks. Rachitis, adenoids, and a neurotic disposition are among the predisposing causes. In some cases the attacks appear to be produced by acute indigestion, the result of errors in diet.

Pathological Anatomy.—The lesions are those of a simple catarrhal laryngitis more or less developed, together with spasm chiefly of the adductor muscles of the larynx. The increasing congestion due to the recumbent position at night, and also the drying of mucus upon the membrane at this time, are assigned as causes for the tendency for the attacks to be nocturnal.

Symptoms.—The attack is often preceded by some hours of moderate coryza and hoarseness, the latter perhaps coming on only in the later afternoon; or the child may have been put to bed apparently entirely well. In still other cases there had been decided laryngitis with fever and a somewhat croupy cough. Usually early in the night, either after gradually increasing slight dyspnea and some cough or quite suddenly, the child wakens with a characteristic barking, metallic cough and noisy stenotic inspiration. It sits up in bed struggling for breath and evidently frightened, and often grasps at the throat where the feeling of oppression and impending suffocation is located. The face is congested and has an anxious expression; the lips and extremities are cyanotic; the alæ of the nose, the sternocleidomastoid muscles, the thoracic interspaces, the episternal notch and the epigastrium move with each noisy, stridulous inspiration. The voice is hoarse; the pulse accelerated; the skin is perspiring; there is moderate or no elevation of temperature and more or less prostration. The dyspnea is not uniformly severe, but increases with any excitement or without discoverable reason. After several hours, or a much shorter time, the severity of the spasm gradually diminishes, respiration grows easier, cough lessens and loosens, and the child sleeps, although some degree of stenosis may still remain, and the attack may recur later in the night. By the following morning no evidence of stenosis is present and perhaps only slight hoarseness and loose cough; or the child seems quite well, and even these symptoms may be absent.

This is the description of the condition as seen in severe cases. In the milder ones there may be a croupy cough and a moderate degree of stenosis, sufficient to arouse the child, but there is no decided dyspnea. In still others the symptoms are not severe enough to make the patient waken.

Course and Prognosis.—In the well-developed cases, such as described, another attack is liable to occur upon the next night unless prevented by treatment, preceded in the afternoon by symptoms similar to those of the preceding day; and still another may take place upon the third night, or even rarely upon later ones. These later seizures are, however, usually less severe, and in the milder cases an attack occurs generally upon one night only. During the daytime the child is apparently in perfect health or has perhaps slight hoarseness and a loose cough which may persist for several days. Although the disease appears so threatening the prognosis is good. If the diagnosis is correctly made, one may expect recovery in practically every uncomplicated case. Fatal instances reported are probably to be classified in the next variety of laryngitis or were dependent upon a pseudomembranous inflammation.

Diagnosis.—The disease is to be differentiated especially from pseudomembranous laryngitis, subglottic laryngitis, and laryngismus stridulus. From *diphtheritic laryngitis*, the usual pseudomembranous form, it is to be distinguished by the very abrupt onset, the short duration of the paroxysms, the usual prompt relief under treatment, the cessation of symptoms during the day, the absence of constitutional involvement, and the history of former occurrences of spasmodic croup. It is only when one is called at the height of the first attack that the diagnosis between the two diseases is uncertain. In true croup (diphtheritic laryngitis) the onset is gradual, the hoarseness and stenosis slowly becoming worse; there is more constantly fever and decided depression of strength, and the cough is not so barking. Especially is to be noted that in most instances diphtheritic croup is accompanied by evidence of diphtheria in the pharynx or nose. *Laryngismus stridulus* exhibits no fever whatever and no hoarseness or cough. The attacks are of momentary duration and are repeated many times during the daytime. Moreover, this is a spasmophilic disorder confined almost entirely to infancy and is often associated with symptoms of rickets or of tetany. The diagnosis from *subglottic laryngitis* will be considered under that heading.

Treatment.—When a child known to be subject to attacks of spasmodic croup develops hoarseness or a suspicious cough during the daytime, an attempt must be made to prevent the nocturnal paroxysm. The same is true during several days after an attack, lest a repetition occur. For this purpose a combination of ipecacuanha and opium may be given, using enough of the former to bring the patient to the verge of vomiting and enough of the latter to cause a moderate drowsiness. The addition of sodium bromide to the combination is often of service, or antipyrine may be added with benefit. There is no need to administer antimony as was formerly much done. It is a dangerously depressant drug in dosage sufficiently large to be of any real service. The child should be kept in a warm room with the air slightly moist for several days until all threatening symptoms are gone.

To prevent attacks of croup in the future all the hygienic measures should be adopted which have been outlined for the prophylactic treatment of rhinitis, as outdoor life and sleeping in airy rooms, as well as cold sponging to the region of the neck or general cool baths. All possible determining causes, such as rhinitis, adenoids and the like must be sought for and removed, not forgetting the frequent influence of acute indigestion in bringing on croup.

For the relief of an attack already under way measures are indicated which relax the spasm and relieve the congestion. The patient should at once be given an emetic of a teaspoonful of syrup or wine of ipecacuanha and be placed in a warm bath. If emesis does not occur in 20 minutes the dose may be repeated. The relaxing effect of both of these remedies will generally relieve the paroxysm. Warm or cold compresses to the neck are also often of service. An additional useful remedy to insure permanent relief for the balance of the night is the surrounding of the child with water-vapor, often best applied in a croup tent. (See Vol. I, p. 236.) In very urgent cases a slight inhalation of chloroform may be employed. It is rarely if ever that intubation is necessary in undoubted cases of spasmodic laryngitis.

3. ACUTE SUBGLOTTIC LARYNGITIS

(Severe Laryngitis; Severe form of Spasmodic Laryngitis; Inflammatory Edema of the Glottis; Submucous Laryngitis)

Etiology.—This variety of acute laryngitis is of very much less frequent occurrence than the varieties already described. It is usually secondary to some milder form of acute laryngitis; much less often the symptoms develop so rapidly that the condition is probably primary. It occurs chiefly in infancy and early childhood. Infectious diseases, especially measles, may give rise to it.

Pathological Anatomy.—There is present the catarrhal inflammation characteristic of simple acute laryngitis but with an increased degree of swelling and of redness. The epiglottis and the ary-epiglottic folds and arytenoid cartilages are swollen and red; the vocal cords may be normal or injected. Usually the most prominent lesion, however, is the swollen, deep-red subglottic mucous membrane, which is seen projecting beneath the vocal cords and almost obstructing respiration. Thickened, drying mucus upon this projecting portion increases still further the stenosis. The inflammation often involves the connective and lymphoid tissue of the submucous layer; and even the perichondrium may be congested. The mucous membrane of the trachea may be seen with the laryngoscope to be for some distance more or less involved in the process.

Symptoms.—The symptoms of acute rhinitis or of mild simple laryngitis may be present for several days, or the attack may start with vigor almost from the beginning, or may be consecutive to an attack of croup. In well-marked cases there is fever up to 103° or 104°F. (39.4° or 40°C.), loss of appetite, thirst, prostration, rapid pulse, hoarseness, and a persistent, painful, hoarse, croupy cough. There is also tenderness and pain in the larynx and a constant annoying tickling sensation there. The degree of dyspnea varies with the case, in some instances being most of the time moderate; in others becoming extreme, with all the attendant symptoms of suffocation as seen in spasmodic laryngitis, but far more persistent. The dyspnea may be paroxysmal to a certain extent, or gradually increase from the beginning and closely resemble the condition seen in diphtheritic laryngitis. Even when decidedly paroxysmal, there is between times only partial relief; and, unlike spasmodic laryngitis, the stenosis is present in the daytime as well, although usually worse at night. There is practically always an accompanying tracheitis, and not infrequently bronchitis follows extension of the inflammation downward, or bronchopneumonia develops.

Course and Prognosis.—Severe symptoms may last for 2 or 3 days or even occasionally a week or more before decided improvement begins. Then in favorable cases they gradually abate, although varying in intensity and showing increase of stenosis, cyanosis, and other signs of the disease from time to time. Complete convalescence may require from 2 to 3 weeks. In other cases the stenosis persists and the child dies in a few days from obstruction to the respiration, or finally perishes from a complicating pneumonia. In 1 very unusual case under my observation,[1] which seemed to belong more to this affection than any other, symptoms lasted for 2½ months, during a portion of which the wearing of an intubation tube was necessary. The prognosis is on the whole favorable, but in the individual case is often most doubtful. The disorder occurring in the course of the acute infectious fevers, notably measles, is very often fatal.

Diagnosis.—Early in the attack the malady may be supposed to be spasmodic laryngitis. It is distinguished, however, by the persistence of the stenosis to some extent during the daytime, the greater duration of the whole attack, the more decided elevation of temperature, and the general constitutional disturbance. In very rapidly fatal cases the diagnosis from spasmodic croup can be made only at autopsy, except that the fact that the attack did end fatally is in itself practically certain evidence that something other than spasmodic laryngitis was present. Occasionally pressure by an enlarged thymus gland may produce a stenosis which bears some resemblance to that of subglottic laryngitis. The slower onset, the more protracted course, the absence of fever and hoarseness, and the examination by the *x*-ray will usually make the diagnosis clear. The differentiation from diphtheritic laryngitis is the most difficult, unless evidences of diphtheria of the nose or pharynx are discoverable, or albuminuria is present. The onset is very similar in each. In subglottic laryngitis, however, there is a greater tendency to paroxysmal increase of stenosis, and less often the steady augmentation of the dyspnea from a gradual beginning as seen in diphtheria. Inspiration is more affected than expiration. The condition is usually worse during the nighttime and the fever is often higher than in diphtheria. To all this there are, however, so many exceptions, and the symptoms may be so like those of diphtheria, that diagnosis may be impossible except by bacteriological and laryngoscopic examination. The latter is often very unsatisfactory in infancy and even a negative bacteriological finding is not an absolute proof, although a very probable one. The history of previous attacks, if these have occurred, is useful in this connection.

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[1] Arch. of Ped., 1902, XIX, 183.

Treatment.—Prompt treatment should be employed to abate the laryngeal inflammation. A purgative should be given if the case is seen early, and a hot bath be followed by putting the child to bed in blankets in the attempt to induce free perspiration. The repetition of hot baths or hot packs during the attack often gives great relief to the dyspnea. Hot, wet compresses over the larynx are frequently of service. Internally the administration of ipecacuanha and opium is useful as in spasmodic laryngitis; combined often with citrate of potash and tincture of aconite root. Antipyrine and the bromides may also be of service if the element of spasm is decided. Of all remedies, however, the inhalation of water-vapor is the most useful. The whole room may well be filled with the vapor; but as this is often difficult in an apartment of considerable size, the employment of a croup-tent is to be recommended (Vol. I, p. 236). The child should be kept in the tent for 20 minutes at a time and the treatment repeated every hour or less often according to the requirements and the effect produced. Too long continuance in the tent at one time is sometimes exhausting, and there is also often an insufficient supply of fresh air. In the severer cases inhalation of oxygen may be combined with the croup-tent treatment. Water alone may be used in the croup-kettle or there may be added with advantage benzoin, menthol or turpentine. When the child is out of the tent the air of he room should be kept as moist as possible by water-vapor medicated as described.

In the severest instances intubation may be required, even though the disease has been proven not to be diphtheria. Inasmuch as the diagnosis is so often doubtful the administration of diphtheria antitoxin should be employed in all threatening cases.

4. PSEUDOMEMBRANOUS LARYNGITIS

This is almost invariably of diphtheritic nature and has already been described. Rarely cases are reported in which no evidence of diphtheria can be found. This condition may be a primary affection of the larynx, or secondary to some of the infectious fevers; or may be produced by irritating vapors or liquids. Such germs as the streptococcus or pneumococcus or others may be discovered in place of the diphtheria bacillus.

The symptoms do not differ from those of diphtheritic laryngitis, and every case of pseudomembranous laryngitis must be considered diphtheritic until proven not to be such.

CHRONIC LARYNGITIS

Etiology.—This disorder is a comparatively uncommon one in early life. It may be a sequel to acute laryngitis; may be primary; may remain a symptom for some weeks after such affections as measles; or may attend a chronic pharyngitis or rhinitis. Adenoid vegetations of the nasopharynx and hypertrophic rhinitis may be accompanied by hoarseness lasting for months. In infancy hereditary syphilis is not rarely the cause of a chronic laryngitis. Of 76 cases of syphilitic laryngitis collected by J. MacKenzie[1] 53 were in infants under 1 year, and 43 of these in the first 6 months of life. Very much more rarely the disease is seen later in childhood as a late symptom of hereditary syphilis. Strauss[2] could find but 14 reported instances of this condition in addition to 3 recorded by himself. Tuberculosis is an unusual cause of chronic laryngitis in early life, especially in infancy and early childhood. Barthez and Sannée[1] observed no cases younger than 3 years in 16 instances of chronic tuberculous laryngitis in children. Only 4 were under 7 years of age. A case is recorded by Heubner[2] in a female infant of 5 months; Rheindorff[3] observed one developing at the age of 8 months, and confirmed by autopsy at 13 months, and Demme[4] saw the disease in a girl of 2½ years. He reports 7 cases in a total of 36,148 children examined.

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[1] Amer. Jour. Med. Sci., 1880, LXXX, 321.
[2] Arch. f. Kinderh., 1892, XIV, 312.

Pathological Anatomy.—This depends largely upon the nature of the cause. In *simple* chronic laryngitis there is moderate redness and swelling of the mucous membrane, but to a less-marked degree than in acute cases. The condition may be general or partial, and the affected regions are coated with mucus or muco-pus, either thin or in process of drying. As an early *syphilitic* lesion there is generally only the evidence of a catarrhal inflammation found, but in some instances the lesions are more deeply situated; and as a later manifestation may be more severe, and ulcerated gummata or mucous placques may be discovered. The epiglottis is swollen and the thick edges rolled in, and inflammation of the cartilage is present. The ary-epiglottic folds show similar changes perhaps with papillomatous outgrowths and the interior of the larynx may be affected, especially the posterior wall. In chronic laryngitis of *tuberculous* nature any part of the larynx may exhibit infiltration, swelling and finally ulceration.

Symptoms.—The chief manifestation is a chronic hoarseness perhaps with a severe laryngeal cough. In tuberculous cases there may be muco-purulent expectoration together with blood and containing tubercle bacilli. Evidences of pulmonary tuberculosis are nearly always present. In syphilitic laryngitis, in addition to the hoarseness and cough, there may occasionally be some degree of stenosis or sometimes severe suffocative attacks, and the symptoms of syphilis are discoverable elsewhere in the body.

Course and Prognosis.—The prognosis of chronic laryngitis dependent upon syphilis is to an extent unfavorable through the possibility of fatal stenosis occurring, and on account of the advanced condition of ulceration and cicatrization which may be present before treatment is commenced. If treatment is begun early, recovery may eventually take place, and this is certainly true of the laryngitis of a catarrhal nature which occurs as an early manifestation of hereditary syphilis. The late forms of syphilis of the larynx are very resistant to treatment. Tuberculous ulceration of the larynx is so overshadowed by the advanced pulmonary disease usually present that death will probably result from this latter cause. Simple chronic catarrhal laryngitis will usually disappear under treatment; which, however, must include the removal of such chronic causes as adenoid growths and chronic rhinitis or pharyngitis.

Diagnosis.—The diagnosis between tuberculous and syphilitic ulceration is not easy by laryngoscopic examination. The symptoms of one or the other of the two disorders elsewhere in the body serve to differentiate. With neither of these diseases present, the recognition of chronic laryngitis and its differentiation from the hoarseness due to papillomata or other cause is to be made by the laryngoscope. The satisfactory employment of the instrument, is, however, difficult in early childhood.

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[1] Mal. des enf., 1891, III, 1053.

[2] Lehrb. d. Kinderh., 1911, II, 245.

[3] Jahrb. f. Kinderh., 1893, XXXIII, 71.

[4] 20 Bericht ü. d. Thätigkeit des Jenner'schen Kinderspitals in Bern, 1882; Ref., Rheindorff.

Treatment.—This should be directed especially against the primary cause. Spraying with weak astringent solutions such as tannic acid may be employed, or the inhalation of water-vapor containing eucalyptus, oil of pinus sylvestris, terebene, or the like; or counter-irritation used externally over the larynx. But these procedures are seldom necessary and often inefficacious, while the cure of the pharyngitis or the rhinitis, the removal of adenoids, or the instituting of careful hygienic measures intended to prevent repeated attacks of acute laryngitis will usually suffice. Syphilitic laryngitis is to be treated especially by the constitutional medication for this disease. If stenosis occurs intubation is indicated.

PERICHONDRITIS OF THE LARYNX

Etiology.—This disease, uncommon at any time of life, depends oftenest upon the acute infectious diseases. It is also caused by syphilis, tuberculosis and sepsis, and may be the result of injury by intubation, or trauma of other nature.

Pathological Anatomy.—The inflammation may attack any of the cartilaginous structures of the larynx, but oftenest the arytenoid cartilages and the cricoid cartilage. Laryngoscopic examination shows redness and swelling and the evidences of an abscess (*Suppurative perichondritis*) or of necrotic cartilaginous tissue; or the process may assume a more chronic adhesive form with the production of connective tissue.

Symptoms.—The onset is insidious. The region of the larynx is tender on pressure and painful. Difficulty in swallowing is usually present. There is hoarseness, stridulous cough, and sometimes dyspnea. The temperature is usually high, but if the condition is secondary to one of the acute febrile infectious diseases this symptom is equivocal, except that the onset of the laryngeal involvement may occasion an increase in the fever already present. If an abscess forms and points within the larynx there is a steady increase of dyspnea, until respiration is entirely obstructed unless relief is given. If it points externally it is readily recognized by inspection and palpation.

Course and Prognosis.—The course will be rapid if the condition is an acute pyogenic process; otherwise it may be slow, especially if no abscess develops. It is chronic in tuberculosis and syphilis. The prognosis is always serious on account of the danger of suffocation often present from the obstruction by an abscess or by acute edema. Deformity of the larynx from cicatricial processes is liable to remain, with permanent alteration of voice or interference with respiration. The prognosis is best in syphilis since the course is slower and the disease often more amenable to treatment.

Diagnosis.—This can be made only with difficulty and sometimes only provisionally. The formation of abscess within the larynx depends oftenest upon perichondritis. A permanent deformity of the larynx after recovery indicates cartilaginous involvement. Abscess pointing upon the outside may be the result of perichondritis or of some other cause. The nature of the primary cause and the study of the other symptoms present will aid in forming an opinion.

Treatment.—Local applications are indicated to reduc
such as ice-bags to the neck, leeching and the like. Any ab
ing must be opened. In cases depending upon syphilis,
stitutional treatment is required. Tracheotomy or intu
necessary to avoid suffocation.

EDEMA OF THE GLOTTIS

Under this heading are frequently included two fo
distinct from each other, the *serous* and the *inflammatory* c
The latter is closely allied to and shades into the subg
already described, and differs from it chiefly in the gre
severity of the inflammation. The serous form is less often seen than the inflammatory in early life, and both are uncommon.

Etiology.—The *serous* variety of edema of the glottis depends upon constitutional causes, especially acute or chronic nephritis. It may also be caused by diseases of the heart, the pressure by tumors, or any other condition which interferes with the circulation in the larynx; angioneurotic edema; or the ingestion of potassium iodide. The *inflammatory* edema is secondary to catarrhal or ulcerating inflammation of the larynx or of neighboring parts. Erysipelas or abscess in the neck, retropharyngeal abscess, and the trauma produced by foreign bodies or by the entrance into the larynx of steam or other irritating or corrosive substances are among the causes.

Pathological Anatomy.—In the *serous* form there is an infiltration of serum into the submucous tissue, the condition being merely dropsical. The ary-epiglottic folds, the epiglottis, and in general the structures forming the upper portion of the larynx are the regions most involved, although any portion may be affected. The epiglottis is greatly thickened and distended and forms a mass easily seen by the laryngoscope or felt by the finger. The ary-epiglottic folds form stiff projecting swellings which may be seen or may be felt by the finger and which may almost or completely close the larynx. The tissues of the trachea may exhibit similar submucous infiltration and swelling. In cases of serous laryngeal edema the parts are pale red. In the *inflammatory* cases the color is deep red and the vocal cords may also exhibit congestion. In addition to the serous effusion a small-celled infiltration is present.

Symptoms.—The clinical manifestations are much the same in each variety. There occurs a more or less sudden inspiratory laryngeal dyspnea with the general characteristics already described in considering spasmodic laryngitis. This is especially true of the serous variety, and a sudden suffocative attack may be the earliest manifestation of a general edematous condition of the body about to occur in chronic nephritis. There is no fever in the serous form unless dependent upon the primary disease. In the inflammatory variety there is pain and swelling, tenderness on pressure, high fever, cough, hoarseness, often difficulty in swallowing, enlargement of the cervical lymphatic glands, and severe constitutional symptoms.

Course and Prognosis.—Apart from the danger of death in a few hours, especially present in the serous variety, the course and prognosis depend upon the nature of the cause of the edema. The majority of cases recover, but the prognosis is always doubtful, and in inflammatory cases recovery may be only after days of illness. In the cases of this nature there is also the danger of the development of bronchopneumonia.

Diagnosis.—In either form the diagnosis can usually be made by exploration with the finger or the laryngoscope, the great swelling of the laryngeal tissue being characteristic of the disease. Even the mere depression of the tongue with the finger may reveal this condition. The slight degree of any redness seen, with the presence of symptoms of nephritis as shown by the urine, denote a serous infiltration. Great redness, with the other symptoms of laryngeal inflammation described, together with involvement of the tissues of the pharynx, indicate the inflammatory form of edema. The differentiation from diphtheria is not always easy. However, in this latter disease there is usually a slower onset and evidences of diphtheritic membrane in the nose or pharynx.

Treatment.—Primarily this should be of the cause. In edema depending upon nephritis energetic measures must be employed to promote diaphoresis and diuresis. (See Nephritis, p. 181.) In inflammatory cases localized antiphlogistic measures are required such as leeching of the neck, the application of ice over the larynx, and the like. Purgatives may also be employed, remembering, however, that the strength is already often much depleted and must be sustained. Scarification of the larynx is recommended but is difficult to perform satisfactorily and is usually ineffectual. If suffocation is impending tracheotomy must be done, as the great swelling of the tissues renders intubation difficult and often dangerous from the trauma produced.

MORBID GROWTHS OF THE LARYNX

Malignant growths of the larynx are very uncommon in early life. Occasionally as a result of chronic laryngitis small fibromatous nodules of a benign nature develop on the vocal cords and produce a roughening of the voice. Small granulomata may follow intubation and tracheotomy. Omitting further consideration of these, the most common morbid growth of the larynx is the papilloma.

Papilloma of the Larynx.—This may be congenital, or be the result of repeated laryngitis. It may occur at any period in childhood, but becomes manifest oftenest between 3 and 4 years of age and is seen more frequently in boys. The growths may vary in size, are whitish in color, may be sessile or have a peduncle, and may occupy any part of the larynx, but usually are attached to the vocal cords. The tumors are usually multiple. The **symptoms** are chiefly hoarseness or even loss of voice, and a paroxysmal cough with more or less stenosis and dyspnœa if the tumors reach a considerable size, or if a catarrhal condition is present. The rapidity of growth is very variable, but usually slow. Sometimes the neoplasms retrograde of themselves; in other cases they require removal. Recurrence is very liable to take place. Sudden death from suffocation may occur. The **diagnosis** is to be made only by laryngoscopic examination, which serves to distinguish the condition from chronic laryngitis of any form. The **treatment** is entirely operative and should be in the hands of a specialist. It is a disputed question whether the growths should be removed, or be let alone and tracheotomy be performed if the symptoms are threatening.

FOREIGN BODIES IN THE LARYNX

Etiology.—The penetration of foreign bodies into the larynx is a not uncommon occurrence in early life. They generally consist of any of the small objects which children are liable to put into the mouth, as well as of articles of food which are being masticated. Fortunately the rapid closure by the epiglottis of the opening into the larynx renders the accident far less frequent than it would otherwise be. It is especially liable to occur during the sudden, deep inspiration attending crying, coughing, sneezing or laughing, and it may follow operative procedures, as, for instance, the discharge of pus after the opening of a retropharyngeal abscess, or the dropping into the larynx of an excised tonsil. Exceptionally bodies may enter the larynx from other regions than the mouth, as in the cases where ascarides have made their way into it.

Symptoms.—The chief symptom of the accident is a sudden attack of suffocation, with violent laryngeal cough. Fortunately in the majority of instances the foreign substance is immediately driven out by the cough, although the sense of irritation may not disappear so promptly. If the object is of sufficient size and is not at once expelled, death may immediately follow from suffocation. If this accident does not happen and the object is not displaced, paroxysmal cough persists, and there are suffocative attacks, bloody expectoration, hoarseness, pain, and other evidences of severe acute laryngitis. If the object is small and smooth the acute symptoms may subside and only hoarseness and moderate paroxysmal cough remain, but at any time an acute fatal attack of suffocation may occur through closing of the glottis by the foreign body, aided by the swelling of the mucous membrane which has taken place. Should the body remain sufficiently long in the larynx ulceration is liable to follow, perhaps with hemorrhage as a result of erosion of vessels.

Prognosis.—This is uncertain, and is in accord with the character, size and position of the object. Ultimate recovery depends upon the possibility of the removal of the object. After its long presence with ulceration, cicatrices develop which may cause persistent stenosis to a greater or less degree. In many cases the object passes from the larynx into a bronchus and occasions a pneumonia.

Diagnosis.—The diagnosis is usually easy. The suddenness and severity of the attack and the absence of fever are characteristic. The laryngoscope may sometimes be of service in older children, but is usually of little value in younger ones, owing to the resistance offered by the patient and the consequent likelihood of increasing the difficulty in breathing and the danger of suffocation. Sometimes digital exploration is effective, although it possesses the same objections.

Treatment.—At the onset this consists in inverting the child in the hope of aiding cough to expel the body. If suffocation is impending the only procedure is immediate tracheotomy. Should the case not be so urgent and the object be still present surgical aid in the removal must be sought at once. The employment of radiography may aid in determining the exact location of the object. The method to be employed in the removal of the body will depend upon its position.

CONGENITAL LARYNGEAL STRIDOR

The nature of this rather uncommon condition has been much discussed. Although described earlier, it was first brought prominently into attention by Lees.[1]

Etiology and Pathology.—It occurs in the new born or in early infancy. The direct cause has not as yet been determined. The theory of Lees and others, based upon post-mortem examinations, is that there exists a congenital alteration of the shape of the larynx, perhaps due to a degree of atony, in which the edges of the epiglottis as well as the ary-epiglottic ligaments approach each other too closely, leaving only a rhomboidal or slit-like fissure at the entrance of the glottis, with consequent obstruction to inspiration. Thomson and Turner[1] attributed it to an incoördination of the respiratory muscles, producing a choreiform respiratory spasm, as a result of which the larynx undergoes a temporary deformity. According to either view the deformity is the same, and the stridor is produced by the vibration of the ary-epiglottic folds during inspiration. Hochsinger,[2] on the other hand, maintained that the disease is not at all laryngeal but due to the pressure of an enlarged thymus upon the trachea; and claimed to have demonstrated this by radiographic methods. This view is so opposed to others that further confirmation is required. It is probable that more than one cause may be operative. Brecelj[3] has seen the symptom attend a pseudodiphtheritic involvement of the palate; Shukowsky[4] has observed it with cleft palate, and Wernstedt[5] at an autopsy on a case dying from another affection could find no deformity of the larynx whatever, and believed that, at least in this instance, the anomaly was a functional one.

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[1] Trans. Path. Soc. of London, 1883, XXXIV, 19.

Symptoms.—These appear at birth or oftener within a few weeks and consist chiefly of a noisy inspiration which varies in intensity from time to time and is made worse by excitement. It is more or less continuous, is always less when the respiration is quiet, and there are times in the milder cases when it is temporarily absent. It may entirely or nearly disappear during sleep. The sound does not resemble that of spasmodic croup, but suggests, according to Thomson,[6] that made by an excited hen. Expiration is generally entirely quiet and there is no urgent dyspnea, although some sinking in of the episternal fossa, intercostal spaces, and of the epigastrium is present. There is, as a rule, no cyanosis, the voice is unaffected, and cough is uncharacteristic, if it occurs. The general health of the infant is usually good or there may be moderate pallor and malnutrition.

Course and Prognosis.—The prognosis is usually favorable. The symptoms persist or even increase in severity for several months, and, although causing great uneasiness to the parent and even to the attending physician, gradually disappear entirely between the ages of 1 and 2 years. Occasionally, however, death may occur, but this is rare and generally from some complication. Should diphtheria, pneumonia, or other disease involving respiration chance to develop, the course of this is rendered much less favorable.

Diagnosis.—This is not always easy. Other conditions causing stridor must be excluded. The absence of cyanosis is said to be characteristic, but I have seen 1 very persistent case, in which the symptoms finally disappeared completely, where the cyanosis was sufficiently marked to occasion fear of asphyxia. Croup is a disease of later infancy or childhood and is sudden in onset and of short duration. Papilloma of the larynx produces hoarseness, and develops at a later period of life. Adenoid growths occurring in very early infancy may give rise to diagnostic difficulty. The noisy breathing in this condition is, however, worse during sleep, and there is evident interference with nasal respiration present. If the congenital laryngeal stridor is indeed the result of malformation of the larynx, this is to be distinguished from the stridor due to pressure upon the trachea by an enlarged thymus gland. The differentiation is difficult or impossible, for even the discovery by radiographic methods of enlargement of this gland does not constitute a proof that a stenotic pressure exists.

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[1] Edin. Med. Jour., 1892, XXXVIII, 1, 205; Brit. Med. Jour., 1900, II, 1561.

[2] Wien. med. Woch., 1903, LIII, 2106.

[3] Jahrb. f. Kinderh., 1904, LIX, 54.

[4] Jahrb. f. Kinderh., 1911, LXXIII, 459.

[5] Nord. Med. Ark., II, H. 1–4, No. 22. Ref., Monatsschr. f. Kinderh., Referat. 1915, XIV, 397.

[6] *Loc. cit.*

In view of the favorable prognosis, **treatment** is scarcely required, other than the effort to overcome any malnutrition present.

STENOSIS OF THE LARYNX

In the broader sense this constitutes a narrowing of the larynx from any cause, and the number of these is considerable. Of those acting within the larynx itself may be mentioned pseudomembranous laryngitis; edema of the glottis; subglottic laryngitis; foreign bodies in the larynx; the alteration in shape seen in congenital laryngeal stridor; very rarely malformations of the larynx of other sorts; syphilitic or tuberculous ulceration; and spasm of any kind, as in spasmodic croup and laryngospasm, and the rare cases of spasm from other causes, as in some cases of apnea from spasm of the larynx occurring in the new born. Produced by causes acting outside of the larynx, stenosis may follow pressure by enlargement of the thyroid gland; abscess; greatly enlarged lymphatic glands, and tumors.

The **symptoms** have been sufficiently considered in describing the various diseases in which passage of air through the larynx is interfered with. They consist briefly of inspiratory dyspnea, with the energetic action of the respiratory muscles naturally resulting; cyanosis; and in some cases suffocation. It is to be distinguished from tracheal stenosis by the altered voice, and the characteristic cough often present. The history of the case and the presence of other symptoms are frequently of aid in recognizing the condition, as they are also in determining the nature of the cause. The treatment depends upon the etiological factors operating.

CHAPTER IV

DISEASES OF THE TRACHEA, BRONCHI AND LUNGS

FOREIGN BODIES IN THE TRACHEA AND BRONCHI

A foreign body entering the larynx may either be expelled immediately, remain impacted, or pass into the trachea or into one of the bronchi, usually the right. The causes of entrance of bodies into the larynx have already been discussed.

Symptoms.—A foreign body in the trachea may promptly cause death if of sufficient size to interfere seriously with respiration. If smaller it may remain in position an indefinite time, producing more or less stenotic dyspnea, a characteristic sound on auscultation caused by the slight moving up and down of the body, and attacks of violent coughing and dyspnea during one of which the object may perhaps be driven upward against the glottis and cause suffocation, or even through it into the mouth and thus be gotten rid of. If the body is immovable there are no paroxysmal suffocative attacks. Ulceration may occur and produce sanguino-purulent expectoration and finally cicatricial contraction with increasing stenosis.

If the object enters a large bronchus the lung supplied by it soon collapses if the obstruction is complete. There is cyanosis and great dyspnea, with violent attacks of coughing, and with pain in the chest. The physical signs consist in the absence of the breath-sounds over the whole lung, or a large portion of it, although early in the condition the percussion resonance is unimpaired. Later there is perhaps dullness, or hyperresonance from emphysema of the neighboring pulmonary tissue, and still later tympany from the production of an abscess cavity. In partial obstruction there is likewise dyspnea, and auscultation may perhaps give a loud whistling respiratory murmur on the affected side with feeble respiration, and there is somewhat diminished vocal fremitus with normal percussion sounds.

Course and Prognosis.—The course is variable and the prognosis is always grave. Escaping the primary danger of immediate suffocation, there is always that of this occurring in some attack of coughing and dyspnea, if the object is loose in the trachea. Later, if the body is in a bronchus, ulceration takes place during a period of quiescence, and there is great danger of the development of symptoms of recurrent or chronic bronchopneumonia, or those of bronchiectasis or pulmonary abscess. In the last there is fever of a hectic type, and great impairment of the general health. Sometimes bronchopneumonia develops in a day or two after the entrance of the body and rapidly advances to a fatal septic condition. Yet a patient may live for months with abscess or bronchiectasis due to a foreign body in a bronchus, and in fortunate cases the object be finally ejected and recovery take place. The general outcome is, however, unfavorable unless the object can be removed.

Diagnosis.—This is generally easy if the history of the entrance of a foreign body into the respiratory tract is known. Without this knowledge diagnosis is difficult or may be impossible. Among the most suggestive symptoms are the sudden onset and the later recurrence of dyspnea and violent paroxysmal cough, and the absence of breath sounds on one side of a bronchus is involved. The employment of the *x*-ray or of the bronchoscope may give conclusive evidence. Later in the course the development of the symptoms or physical signs of abscess, or a chronically recurring bronchopneumonia especially in the right lower lobe. is a suspicious indication of a foreign body.

Treatment.—Treatment can be surgical only. The inversion of the patient combined with striking him upon the back is a dangerous procedure if an object has passed below the larynx, lest it be thus made to obstruct the glottis from below. Tracheotomy should be first performed or all preparations for it made. The employment of the bronchoscope has rendered the removal of foreign bodies more readily possible than was formerly the case.

STENOSIS OF THE TRACHEA AND LARGER BRONCHI

This is a result of various **causes,** and may even rarely be congenital. Of the conditions situated within the tube the most frequent is diphtheritic inflammation, which may produce pseudomembrane and consequent stenosis, as may also the fibrinous bronchitis which will be considered later (p. 38). The action of foreign bodies has just been described. In the walls of the tube itself are to be enumerated as causes strictures of various sorts, such as those depending upon cicatricial growths after tracheotomy, or resulting from syphilitic or other ulceration. Compression of the trachea or bronchus from without is a cause of another sort. Here is to be mentioned pressure by an enlarged thymus, a considerable number of undoubted cases of which have been reported (see Diseases of the Thymus, p. 520), by hypertrophied bronchial and mediastinal glands, mediastinal tumors, an enlarged thyroid gland, and retroesophageal or other abscess in the neck or within the thorax.

The **symptoms** may develop suddenly or slowly, according to the nature of the cause. They consist in dyspnea especially during inspiration, which is often attended by a peculiar characteristic noisy whistling sound; often more or less cyanosis; and the vigorous action of the respiratory muscles as seen in dyspnea from any cause. Cough is not infrequent. It is dry, barking and somewhat paroxysmal. When a bronchus is involved the physical signs are those described under Foreign Bodies in the Bronchi (p. 28); namely, feeble respiratory murmur and vocal fremitus, with normal percussion resonance early in the course. In tracheal stenosis the respiration is usually more noisy than when a bronchus is involved, and there is no difference between the physical signs of the two lungs.

Prognosis and treatment depend entirely upon the nature of the cause.

TRACHEAL FISTULÆ

An opening between the trachea and the esophagus is a rare occurrence of congenital origin sometimes seen. It has already been described under Diseases of the Eosphagus (Vol. I, p. 692). Very rarely an opening may form from the trachea into the esophagus as the result of trauma with subsequent ulceration.

ACUTE BRONCHITIS

(Acute Tracheobronchitis)

Tracheitis seldom occurs alone, but is generally combined with laryngitis, or develops with, or is quickly followed by, the involvement of the bronchi as well. In infancy the finer bronchi are especially involved; in later childhood chiefly the trachea and larger bronchi. The varying conditions may be considered together, under the heading "Bronchitis."

Etiology.—The disease is exceedingly frequent and may be either primary or secondary to some other affection. The variety of conditions causing secondary bronchitis is large. Among the infectious diseases producing it are measles, typhoid fever, pertussis, grippe, rubella, diphtheria and scarlet fever. Bronchitis is also a common attendant on or sequel to laryngitis, rhinitis and pharyngitis; is seen often with pneumonia, pleurisy, rickets and malnutrition; and readily develops in patients with cardiac disease and passive congestion of the lungs. Primary acute bronchitis is usually the direct result of chilling brought about by insufficient clothing, the sudden checking of perspiration, high winds, wet feet, and other causes as mentioned under rhinitis. Continued imperfect hygiene, impaired general health from any cause, life in hot, damp, or illy ventilated rooms, and the like are predisposing influences to repeated attacks. Occasionally bronchitis is observed due to the inhalation of irritating substances. The season of the year is a factor of moment, the disease being much more frequent in the cold months, especially, perhaps, autumn and spring, when sudden changes in temperature are most marked. Early age is a powerful predisposing factor. In the first 3 years of life, except the first 6 months, it is one of the most common affections encountered; but after this period it decreases steadily in frequency. The agency of microörganisms is positive but the exact action uncertain. It is likely that under the influence of congestion brought about by local chilling or in other ways, the normal condition of the mucous membrane is so altered that germs of different sorts are able to multiply and produce the symptoms of the disease. Probably the pneumococcus, streptococcus and micrococcus catarrhalis are most active in this connection; but the staphylococcus and the influenza bacillus are also to be mentioned. It is on account of this infectious quality, as in the case of rhinitis, that family outbreaks of bronchitis are not infrequently seen, and it seems probable that during outbreaks the virulence of the germ may be sufficient to produce the disease without the operation of any predisposing factor.

Pathological Anatomy.—The process is diffuse, affecting the bronchi of both sides of the chest, although not uniformly throughout. A bronchitis localized in a portion of one lung is generally secondary to some other pulmonary lesion. In older children the trachea and larger bronchi are involved, except in the severer cases where the middle-sized bronchi share in the process. In young infants there is a greater tendency to inflammation of the smaller and even the smallest bronchioles. When the smallest tubes are attacked, the term "capillary bronchitis" is often employed; but as this process is always accompanied by an inflammation around the bronchioles the disease is in fact a bronchopneumonia. The title "capillary bronchitis" therefore is one to be avoided as tending to cause confusion. In general, the smaller the bronchi affected the more severe is the process. On section of the lung, followed by compression, the secretion can be seen to ooze out of the smaller tubules. In the milder cases the process is confined to the mucous membrane. This is swollen, red, injected, and infiltrated with round cells, and the epithelium is desquamated. The secretion is at first diminished, but the swelling, infiltration and, later, increased secretion of the mucous glands soon coats the surface of the membrane with mucus, which subsequently becomes muco-pus from the addition of cells. In the severer cases the entire thickness of the wall of the bronchi is infiltrated with leucocytes. The bronchial lymph nodes are enlarged, and may be very decidedly so in cases which have lasted for some time. In infancy some of the bronchi may become occluded by the secretion, and collapse of a portion of the lung occur, this being generally combined with emphysema of other portions.

Symptoms.—The symptoms vary very greatly in intensity. In the *mild form* the onset may be acute, or be very gradual, and is very commonly preceded by the symptoms of rhinitis, pharyngitis or laryngitis. Cough begins or, if already present and laryngeal in character, becomes frequent, sometimes paroxysmal, dry, and evidently without production of secretion. It is often more distressing at nighttime and is frequently excessively annoying soon after the child is put to bed. A sense of soreness or discomfort in the chest is often complained of by those of sufficient age. Constitutional involvement of any sort is absent or slight; consisting, perhaps, of loss of appetite or other insignificant digestive disturbances, with little or no elevation of temperature in older children, and only moderate fever of slightly over 100°F. (37.8°C.) in infants. In from 1 to 2 days, as secretion becomes established, the cough grows looser and less racking, and in children over 6 or 7 years of age expectoration begins. This is at first of transparent or whitish mucus only; later it is greenish or yellowish. No expectoration occurs in young children, the secretion being swallowed or reaching only as far as the larynx and returning again to the bronchial tubes. Sometimes vomiting is induced by the violence of the cough, or by the choking caused by the large amount of secretion in the throat.

The physical signs vary with the stage of the disease. At first there are only a few sonorous or sibilant rhonchi audible, or none at all. Later coarse, moist râles can be heard over the entire chest, but especially at the bases behind. If the disease is limited to the trachea and the largest bronchi there may be no râles at all or only rhonchi. The fremitus produced by the râles can often be felt on palpation or heard at a distance from the chest. The percussion note is not affected. It is to be observed that the number of râles, provided these are coarse in character, appears to bear no necessary relationship to the severity of the symptoms in general. Very numerous râles may occur in children with only moderate bronchitis, while an adult with an acute bronchitis exhibiting râles much less numerous would probably suffer from decided constitutional disturbance.

Severe bronchitis owes its character chiefly to the fact that the smaller bronchi are involved. In infancy the fever is higher than in the milder form, reaching 101° to 103° F. (38.3° to 39.4°C.), the cough more constant and distressing, and there are acceleration of respiration, dyspnea, sometimes slight cyanosis, and often more or less prostration. In older children also the onset may be with such decided constitutional symptoms as headache, chilliness, and high fever, and the patient be ill enough to be confined to bed. After secretion becomes established the temperature ceases to remain high in uncomplicated cases.

The physical signs of severe bronchitis are very similar to those of the milder form except for the presence also of fine râles, often in large numbers, in the smaller tubes, combined in infancy with feebleness of the respiratory murmur from the obstruction to the passage of air.

Complications and Sequels.—Bronchitis is oftener itself a secondary disease than a primary affection liable to be attended by other morbid conditions. The complications are those of the respiratory tract. Rhinitis and laryngitis are common at the beginning. Pneumonia is a dangerous complication or sequel especially in infancy. Recurrent attacks of acute bronchitis can readily, under unfavorable conditions, be followed by the chronic form. Tuberculosis of the lungs in older subjects may date its development from an attack of bronchitis.

Course and Prognosis.—The duration of acute bronchitis is from 1 to 2 weeks in the milder cases not occurring in conjunction with other diseases. Although the dry unproductive cough lasts usually only 1 to 2 days, the severity of the coughing may not begin to ameliorate for some time longer. Fever if present lasts only 1 or 2 days. After this, improvement in symptoms with disappearance of râles goes on gradually. The severer cases are prone to last from 2 to 4 weeks before recovery is complete. The fever remains high 2 or 3 days and then may be slight for a few days more. In all cases of bronchitis there is a great disposition to relapse unless precautions are taken; and under unfavorable environment a child may suffer from recurring attacks perhaps for months. The prognosis as to the duration of bronchitis is, therefore, always somewhat uncertain; that as regards recovery is generally good except in severe attacks in infancy. In very many such attacks in infants the process passes rapidly into a pneumonia. In other instances, especially in quit[illegible]g infants, or in those older who are debilitated or rachitic, severe bronchitis may cause death owing to the difficulty experienced in clearing the bronchial tubes of secretion (*Suffocative bronchitis; suffocative catarrh*). The cyanosis then gradually or rapidly increases, the pulse is feeble, the respiration becomes shallow and rapid, there is little cough or crying, the temperature may be normal or subnormal, and the infant dies in a stuporous condition with great prostration, unless relieved in some way. In older children there is generally little danger of this sort, although cases do occur in which death is produced apparently by suffocation by the overwhelming amount of secretion. Usually, however, even in severe cases, profuse secretion, sometimes blood-stained, is established in a few days and removed by coughing.

Many influences affect the duration and the severity of the disease, and the tendency to a fatal ending. In infancy bronchitis is always more threatening than later, owing to the danger of it developing into a bronchopneumonia or atelectasis. Bronchitis secondary to typhoid fever or pertussis runs a longer course than is usual with a primary attack. That associated with measles is often severe but seldom of unusually long duration. In any debilitated condition of the general health bronchitis is liable to recover slowly; and this is especially true of rachitis. In children even of 18 to 24 or more months of age the subjects of decided thoracic deformity from rickets, an attack of severe bronchitis is to be dreaded. Recovery from it is slow, or death may readily occur from the suffocative condition referred to, brought on by the interference with respiration the result of the yielding chest-wall, the feeble cough and respiratory movement, the inability to expel the mucus, and the danger of pulmonary collapse. Failure to protect a child most carefully after bronchitis has commenced, or the continuance of such other imperfect hygienic conditions as have been described as factors in producing the disease, may readily be the cause of constantly recurring attacks or of bronchitis of a subacute form.

Diagnosis.—The disease most difficult of differentiation from bronchitis is bronchopneumonia, and it is probable that many cases supposed to be the former were in reality mild attacks of the latter. In pneumonia the temperature is high to a greater degree and for a longer time, the patient is evidently more ill, the respiration is usually more accelerated and labored, and there are often physical signs of consolidation, especially the presence of fine râles over a limited area, with impairment on percussion or unduly harsh or slightly bronchial respiration here. In general, temperature which continues high for longer than 1 or 2 days, or a rate of respiration decidedly out of proportion to the temperature or the other symptoms, is an indication that something more than bronchitis is present. Râles of moderate size limited to a small area may indicate tuberculous consolidation. Cases with loose cough and no râles or only coarse rhonchi may depend upon tracheitis or bronchitis of the larger tubes, but may, on the other hand, be the result of pharyngitis. The first stage of pertussis is often very confusing. As a rule, early in this disease there are very few if any râles, the cough is more paroxysmal and worse at night, and the condition grows worse in spite of treatment. In some cases of bronchitis, however, the cough is of a very hard paroxysmal character, and no râles are heard, and only the passing of time can distinguish pertussis from a tracheitis or a bronchitis of the large tubes.

Treatment. Prophylaxis.—Prophylactic treatment is of the utmost importance, not only on account of the discomfort from the disease itself, but from the danger in young subjects of the development of bronchopneumonia. All possible causes likely to produce bronchitis must be sought for and avoided, and all hygienic and other measures followed which will tend to strengthen the child and prevent susceptibility to attacks. As these have been discussed at length under the etiology and prophylaxis of Rhinitis (pp. 4, 6), they need not be repeated here, except to emphasize again the importance of keeping a child much of the time in the open air; of avoiding too much or too little clothing and of the playing upon the floor upon cold winter days; of giving cool morning baths, and of sleeping in a well-ventilated bedroom. By this, however, is not meant that every infant can sleep with impunity in a room actually cold. This is largely an individual matter, and delicate infants often do not tolerate it well. In addition, energetic hygienic and medicinal remedies must be at once employed for the cure of rhinitis, pharyngitis or laryngitis already developed, owing to the well-known tendency for the inflammation to spread to the bronchial tubes. This is particularly true where the presence of rickets occasions repeated attacks of bronchitis, or where one has to do with delicate children with tuberculous ancestry. In the latter case, especially, residence in a warmer climate may be of great benefit in the winter season. Tonic remedies are often needed, such as iron, strychnine or cod-liver oil.

Treatment of the Attack.—At the onset the disease may sometimes be aborted by giving a full dose of some purgative medicine, a hot general bath or hot mustard foot bath and a hot lemonade, and covering the patient warmly in bed in the hope of producing sweating. Needless to say the child should be confined to bed in a warm room upon the following day, and as much longer as fever lasts. In some cases the administration of quinine in full doses is efficacious. Should the patient not be seen sufficiently early for efforts at abortive treatment, the remedies employed vary with the age of the patient and the severity and stage of the disease. As regards *internal medication* much can be accomplished, especially in the early stages, by the administration of syrup of ipecacuanha (4 to 8 minims) (0.25 to 0.49) in combination with deodorized tincture of opium (1⁄8 to 1⁄2 m.) (0.008 to 0.032) every 2 to 3 hours at 2 years of age; or, in older children, with heroin or morphine, remembering the susceptibility of infants to opiates, and the possibility of either these or ipecac disturbing digestion at any age. In spite of the apparently opposing action of these drugs, and although there has been much written against the use of each of them, my own experience has often shown prompt relief of the troublesome cough after their exhibition. When secretion is well-established opium is usually contraindicated as it diminishes this and especially the power of emptying the bronchial tubes. It is particularly to be avoided in infants with free secretion and diminished expulsive power. In many severe cases in infancy atropine is often of very decided value, as it is both a stimulant to respiration and a diminisher of secretion. Enough should be given to stop just short of producing flushing of the face—probably 1⁄2000 to 1⁄1000 of a grain (0.00003 to 0.00006) every 3 hours at from 1 to 3 years of age. It should not, of course, be combined with an expectorant. After secretion has begun but has not become excessive in amount, in place of efforts at diminishing it, it is sometimes advantageous to give stimulating expectorants such as squills, chloride of ammonium, terpene hydrate, iodide of potassium, or citrate of potassium, with the intention of favoring and thinning the secretion. In the choice of these two plans one is guided toward the first by the fineness and number of the râles with the presence of dyspnea or perhaps cyanosis; and toward the second by the coarseness of the râles and the absence of any alarming symptoms.

In older children expectorants are oftener indicated, the treatment with atropine being less frequently needed, although useful in some instances. Even in the stage of secretion, however, the cough is sometimes so frequent, disturbing, and unproductive, that more actual benefit is obtained from the administration of opiates, provided no contraindications are present. Considerable judgment is to be exercised, and one should not too readily turn to opiate treatment to allay moderate cough. In many cases of severe frequent cough relief can be secured by the bromides, bromoform or chloral. Apomorphine and antimony are to be avoided for children. In the severest cases where fever is present such remedies as phenacetin may well be added to the treatment. Emetics are seldom needed in bronchitis at any age. Ipecacuanha may often be pushed with advantage to the production of slight nausea, but actual emesis is required only in the unusual cases where the secretion is very abundant and cannot be expelled satisfactorily. I have produced it with advantage in occasional severe cases in older children; but infants with such symptoms are usually too weak to bear emesis well. If there is decided evidence of debility alcoholic stimulants should be employed. In other cases digitalis and strychnine are of value for cardiac weakness.

Yet internal medication for bronchitis at any time of life is always liable to produce secondary effects, especially disturbance of digestion. In infants it may often be dispensed with, and in older children be resorted to with hesitation, if the desired results can be accomplished in other ways.

In all cases *hygienic* treatment is of great value. Older children with fever should be confined to bed; those without fever may be up and dressed, but confined to one room and carefully guarded from draughts. Their afebrile state renders increase of the bronchitis by improper exposure very easy. Playing upon the floor on cold winter days readily prolongs the attack. All infants with bronchitis should be in bed. This does not mean, however, that they can be safely permitted to lie in one position. They should be turned frequently, as well as taken up at times and held in the nurses arms. Indeed, in dangerous cases in very young infants, relief is sometimes obtained by suspending the child for a moment by the feet and allowing the secretion to flow from the mouth. The room, although well ventilated with access of abundance of fresh air, should be of a uniform temperature, slightly higher than in health; about 70° to 72°F. (21.1° to 22.2°C.). This plan of treatment is, I believe, certainly the safest and most satisfactory in the great majority of cases, but like all others, is open to exceptions. I have seen afebrile cases, for instance, where the attack was lasting a longer time than usual, in which prompt improvement followed the exposure of the child to fresh outdoor air near the open window. There can be no absolute rule, and each case must be treated individually in this respect.

Various other remedial measures are often of great service in addition to the internal medication and the hygienic treatment described. The choice of these depends upon the symptoms. *External applications* are often of more benefit than anything else, especially in infancy. Rubbing with amber oil, camphorated oil, or soap liniment, or with a mixture of turpentine and oil is sometimes useful, but often not efficacious or too slow in action. The best method, especially in the severer cases, is the application of a homemade mustard-plaster every 3 hours, allowing this to remain in position sufficiently long to produce a redness which will persist after the plaster is removed. The strength of the mustard-paste depends upon the age of the child. (See Vol. I, p. 238.) The wearing of a cotton jacket (p. 244) does not often, in my opinion, serve any good purpose. In those older children who rebel too vigorously against the use of mustard, tincture of iodine may be applied, remembering, however, the susceptibility of young subjects to this and the need often of diluting it. Dry cups are of great aid in many severe cases in older children. In young infants suffering from dangerous respiratory obstruction by the retained secretion, with failing circulation, the use of a strong mustard-pack is sometimes remarkably efficacious. (See Mustard Pack, Vol. I, p. 242.) In other instances where the vital powers are clearly failing the employment of the hot tub bath (Vol. I, p. 240) often does good, although the shock is capable of hastening a fatal ending in those whose cases are hopeless. It should, however, certainly be tried in all grave cases not relieved by other measures described. Its chief action is the rousing of the infant and the consequent crying and deeper respiration which follow. An analogous procedure for the bringing of the blood to the surface, the relief of the stasis of the lungs and heart, and the stimulation of respiration, is the employment of alternating hot and cold douches, or of flagellation, as used in the treatment of atelectasis in the new born.

Inhalations are frequently beneficial. Sometimes improvement does not begin until the room is filled with water-vapor. This may be used alone or medicated with benzoin, turpentine, or menthol. In severe attacks the inhalation may be given in a croup-tent. Where there is cyanosis and dyspnea from retained secretion and insufficient aëration inhalations of oxygen may be employed.

Should cough persist unduly long after other evidences of the disease have disappeared, or should the attack pass into a subacute form, change of climate and the administration of tonic remedies will be of most avail, combined with other treatment suitable for chronic bronchitis.

CHRONIC BRONCHITIS

Etiology.—This not uncommon affection may affect children of any age. It is the result of repeated attacks of acute bronchitis between which the intervals are very short and finally cease entirely. The agencies which tend to produce it are those predisposing to acute bronchitis, especially rickets, the exudative diathesis, general debility, and imperfect hygienic conditions. This last is of very great importance, both among the poor and among those in better circumstances. The living in damp dwellings, or in hot and badly ventilated rooms; the local chilling by draughts after being overheated, and the like, serve to produce a bronchitis which may last the entire winter. Improper feeding is a fertile source in some children, an excess of carbohydrates being the most frequent dietetic fault. Other disorders of the lungs such as asthma, emphysema, tuberculosis and chronic pneumonia are liable to be attended by evidences of bronchitis also. Chronic valvular disease of the heart is another common cause, as is nasal obstruction and consequent mouth-breathing. An occasional danger in measles and a frequent one in pertussis, is the persistence of a chronic bronchitis as a sequel. But apart from all these there is a decided tendency in some children, otherwise healthy, to the constant persistence of slight cough and of a few coarse râles in the chest especially in the winter season. In fact the predisposing influence of the cooler season of the year on the development of chronic bronchitis is very decided.

Pathological Anatomy.—The lesions vary greatly with the case. In the milder instances there is little discoverable except the presence of pus and mucus in the bronchial tubes. In the severer ones the mucous membrane is greyish-red or brownish-red, and there occur a cellular infiltration and increase of connective tissue in the wall of the tube, with more or less dilatation and a fibrinous peribronchitis. Emphysema or atelactasis may be present also.

Symptoms.—There is seldom the fever seen in the acute disease. The general condition is not at all affected in the milder cases and the chief symptom is moderate chronic cough. Instances of this sort occur especially in infancy and early childhood.

In the more prolonged and severer cases, which are commoner in later childhood, the cough is more troublesome, coming on often in paroxysms which may serve to free the bronchial tubes of the secretion which has accumulated. Consequently it is often worse in the morning on awakening. In other cases it is either dry or loose, but without dislodging the mucus, and it is sometimes very distressing during the entire night, and may be sufficiently severe to produce vomiting. The amount of secretion varies considerably. It is usually not profuse unless the disease is complicated by bronchiectasis. Owing to the common custom in early childhood of swallowing the secretion this may seldom be obtainable; yet children suffering with chronic bronchitis often learn to expectorate at a much earlier age than others. It is then seen to be a tenacious mucus or muco-pus. In the severer cases there is often some degree of constitutional involvement represented by anorexia, debility and loss of weight. This is, however, seldom great. There is usually but little dyspnea.

Physical examination shows the presence of coarse râles or rhonchi, which may often be readily heard even without putting the ear to the chest, together with a rhoncial fremitus on palpation; or the râles may temporarily disappear after a paroxysm of coughing, to reappear later; or in other cases nothing abnormal may be found at any time. Chronic bronchitis is liable to vary with the state of the weather. A period of damp weather often exaggerates the symptoms, and the disease is always worse in the winter season. The confinement in school, with the exposure to draughts and other unfavorable influences which often attend this, is another cause for the greater severity during the school year.

Complications.—Most prominent among these are bronchiectasis, which may develop in the severe long-continued cases; atelectasis, seen in young infants and in older ones subject to rickets; and emphysema, which is associated with chronic bronchitis particularly in the long-continued cases, or in those with the tendency to asthma presently to be described.

Course and Prognosis.—The duration is indefinite but nearly always much prolonged. The symptoms may consist in a series of exacerbations with periods of partial improvement; or no improvement at all may occur until the summer season comes on, and the disorder may recur again in force with the advent of autumn, unless successful treatment has been instituted in the meantime. The prognosis is favorable so far as life is concerned, but uncertain as to the duration, although ultimate complete recovery will take place if serious complicating structural pulmonary lesions do not develop, or if no incurable cause is operative, such as chronic cardiac disease.

Diagnosis.—This is not always without difficulty. The disease is to be distinguished from cough depending upon reflex causes, pharyngeal irritation, adenoid growths, and the like. It often much suggests pertussis, which will, however, be excluded as time passes. Tuberculosis of the lungs gives more or less fever, especially in the evening, and a greater constitutional debility. The absence of the cutaneous tuberculin reaction will aid in excluding this disease, but the examination of the sputum for tubercle bacilli may be required to settle the diagnosis in doubtful cases with a positive reaction. Even the discovery of bacilli, however, does not exclude the presence of a chronic bronchitis, since this is so often an attendant upon tuberculosis.

Treatment.—This consists first of all in the removal of the cause. Rickets especially is to be cared for in younger subjects, since serious rachitic deformities of the chest make recovery from severe bronchitis a matter of great difficulty. Valvular disease of the heart requires cardiac tonics and other remedies to restore compensation. Conditions of general debility must receive treatment. For this purpose change of climate to a drier or warmer region, particularly in winter, is one of the best of remedies; or in summer residence at the seashore or in the mountains may give the city child the required tone to overcome the tendency to winter recurrence. Life in the open air is of great value, combined with cool sponging of the body, inasmuch as these measures increase the resisting power; although at the same time clothing of sufficient warmth must be used to prevent surface chilling upon cold days. The importance of diet is not to be forgotten, and in troublesome cases a careful study should be made of the effect of changes in this. The employment of stimulating applications to the chest is also of service; such as rubbing daily with soap liniment, oil of amber, or a combination of sweet oil and turpentine.

Of internal remedies, cod-liver oil is one of the best, with iron or arsenic if there is decided anemia present. For the direct modification of the bronchial mucous membrane most remedies are unsatisfactory. The best appear to be iodide of potassium; the turpentine derivatives such as terebene and terpene hydrate; eucalyptus; creosote; and preparations of tar. Some of these, as eucalyptus, creosote and terebene, as well as benzoin, oil of pine and other similar drugs, may well be given by inhalation, either in water-vapor or nebulized by an atomizer. Opiates are to be used only when the severity of the cough renders this unavoidable.

FIBRINOUS BRONCHITIS

(Plastic Bronchitis)

Etiology.—The most frequent cause of this condition is the extension of a diphtheritic pseudomembranous inflammation from the larynx or trachea. It may also sometimes attend croupous pneumonia, cardiac disease, tuberculosis, or asthma, or be a complication of many of the acute infectious disorders. Apart from these conditions it may very rarely occur as a primary affection, the cause of which is uncertain. It is the non-diphtheritic disorder to which attention is especially given here. Various microörganisms have been associated with it, oftenest the pneumococcus and the streptococcus. The disease may occur at any age. It has been studied with especial care by Bettmann.[1]

[1] Amer. Jour. Med. Sci., 1902, CXXIII, 304

Pathological Anatomy.—The lesion consists in the production of a pseudomembrane in either the larger or smaller bronchial tubes, which is loose or adherent, solid or hollow. Sometimes the deposit is quite extensive and may be expectorated as a dendritic cast of a considerable portion of the bronchial tree; sometimes the casts are in small pieces only. They may be very slender, and so rolled up that their nature is not recognized until they are floated in water.

Symptoms.—The disease appears in two forms, the acute and the chronic. In the *acute form,* a comparatively small group, the onset is as in ordinary bronchitis, but the symptoms are more severe. There is fever, great dyspnea, distressing cough and often cyanosis. Auscultation reveals the presence of râles and often of a fluttering sound if the membrane is loose; but a diagnosis cannot be based on this alone. The important diagnostic signs are the troublesome cough, the severe dyspnea and the expectoration of fibrinous casts. The symptoms are not equally well-marked at all times, but come on in attacks. The expectoration of membranous material greatly relieves the dyspnea and other symptoms, although usually only temporarily so. The duration of the whole process varies from 1 to several weeks; and ends in recovery, or oftener fatally through exhaustion or suffocation. The mortality is high.

In the *chronic form,* very much more frequently seen, there are suddenly developing recurring attacks lasting a few days at a time, with short or long intervals between them, the disease extending possibly over years. The acute constitutional symptoms are absent; the manifestations being cough, dyspnea, and the expectoration of casts composed of mucin. The physical signs are those of chronic bronchitis or of asthma, the disorder possessing points of resemblance to the latter affection. The expulsion of the casts relieves the symptoms. The disease in this form is not usually dangerous to life but is very persistent.

Treatment.—This is very unsatisfactory. Iodide of potassium offers the best hope of cure in the chronic cases. In the acute form effort must be made to obtain expulsion of the pseudomembrane. For this purpose warm sprays of lime-water or of water impregnated with other alkalies or with turpentine, tar, or the like, have been recommended. Emetics are sometimes of value. Pilocarpine may be tried cautiously, but is a dangerous remedy for children. Inhalation of water-vapor may be of service; this treatment being best given in a croup-tent. Unless it has been determined with certainty that the pseudomembrane is not diphtheritic, antitoxin should be administered.

ASTHMA

(Asthmatic Bronchitis. Bronchial Asthma)

Nature.—This disease may show itself in several forms. Its nature is to an extent two-fold; on the one hand a catarrhal bronchitis, and on the other a respiratory neurosis; one or the other predominating according to the case. The subject is, therefore, often considered under two distinct headings, although these are not sharply differentiated: (1) *Asthmatic Bronchitis,* in which the catarrhal element plays a large or the larger part; (2) *Bronchial Asthma,* in which the neurosis is chiefly represented. Regarding the character of the neurosis there are different opinions, attributing the symptoms respectively to spasm of the bronchial muscles, sudden swelling of the bronchial mucous membrane, or rapid secretion into the bronchioles. It is probable also that the nerve-centers are in a state of increased irritability.

Etiology.—The influence of age is very decided. Typical attacks of bronchial asthma are not often seen before the beginning of later childhood, and even in this period are not very common. In 225 cases of the disease tabulated by Salter[1] 11 began in the first year, and 71 in the first 10 years of life. On the other hand, asthmatic bronchitis is not infrequent in early childhood and even in infancy. Heredity plays an important rôle, the parents having suffered from asthma, or in other cases from gouty symptoms or neuroses of various sorts, and I have seen the family tendency shown in twins of 4½ months with a rather severe form of the disease. Obstinate eczema in infancy may be evidence of the exudative diathesis which later manifests itself by asthma. The presence of adenoid growths in the nasopharynx, or the existence of well-marked rickets may be a predisposing cause, and the frequent repetition of attacks of bronchitis tends to be followed eventually by an asthmatic condition.

The immediate *exciting* causes are many. Among them may be mentioned exposure to high wind, dampness, or sudden change of temperature; the occurrence of an attack of bronchitis; the presence of enlarged bronchial lymphatic glands; acute indigestion; auto-intoxication of some sort; the ingestion of certain articles of diet varying with the case, proteins probably being the cause in all such instances; psychic disturbances; and the inhalation of various irritating substances, any one of which contains a protein which may act specifically for the individual case. The relationship to food is especially interesting. The idiosyncrasy to proteins of different sorts (*anaphylaxis*) has repeatedly been observed, even that to cow's milk being seen in occasional instances, while the anaphylaxis produced by egg-albumen is of rather common occurrence. In such cases rubbing the flexor surface of the arm with the food or the extracted protein will produce an urticarial wheal (Talbot;[2] Schloss[3]), and may even produce an attack of asthma sometimes exceedingly severe. At times slight preliminary scarification is necessary. One patient may develop asthma whenever brought anywhere near a horse, another is affected by the presence of other animals, and another by the odor or the pollen of certain flowers and grasses. Sometimes the peculiar reaction of the patient is clearly inherited; in other cases it appears to have been acquired by a gradual sensitization. It is to be borne in mind that only a certain proportion of the cases of asthma can be attributed to the influence of a foreign protein.

Pathological Anatomy.—There are no distinctive lesions found other than those of complicating conditions. The catarrhal element of the disease manifests itself by the changes characteristic of bronchitis, while in long-continued cases more or less persistent emphysema develops.

Symptoms.—The disease is conveniently divided into (1) Asthmatic bronchitis and (2) Bronchial asthma, the two types united by intermediate forms.

1. Asthmatic Bronchitis.—In this form the attack begins as an ordinary bronchitis with cough and fever, but may soon exhibit also distinctive asthmatic symptoms, the element of spasm developing. The fever generally disappears altogether in a few days, but the other symptoms continue. Cough is moderate in severity and expectoration is usually absent, or if obtainable is glairy and tenacious and sometimes abundant. The respiration is decidedly more rapid than normal, the expiration prolonged and often noisy, and there is more or less dyspnea which may reach the stage of orthopnea, especially at night. The general condition is usually good, children wishing to be about and to play. Examination of the chest shows the thorax unduly distended and hyperresonant and the respiratory excursion limited, while on auscultation all sorts of dry, coarse bronchitic râles are audible especially with expiration. The attack may come on with great severity and last only 1 or 2 days, but oftener continues 2 to 4 weeks, the cough lessening, the râles becoming moist, and the asthmatic symptoms gradully disappearing. There may then be an interval of considerable time, sometimes entirely free from symptoms, sometimes showing occasional wheezing and slight shortness of breath from the persistence of the complicating emphysema. Renewed attacks, however, are liable to be brought on by exposure to cold or dampness or by slight digestive disturbance. Very frequently these follow each other with such short intervals that the condition seems to be an almost continuous one, although with exacerbations, and may last for months or during the whole winter, since it is particularly in the winter season that asthmatic bronchitis is most troublesome.

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[1] "On Asthma," 1868, 113.

[2] Bost. Med. and Surg. Journ., 1914, CLXXI, 708.

[3] Trans. Amer. Ped. Soc., 1915, XXVII, 62.

Recurrent Bronchitis.—This is a condition closely allied to and probably but a mild form of asthmatic bronchitis. It resembles ordinary bronchitis in some of its symptoms, but differs in the absence or slight development of fever and of other evidences of general illness, and in the fact that the etiological factors producing it are rather those observed in asthma, especially the ingestion of some food not suited to the patient. Sometimes the usual symptoms of asthmatic bronchitis are associated. The attacks last several days, and may recur every few weeks.

2. Bronchial Asthma.—This is the variety in which the nervous element predominates, although the disease differs from the typical condition seen in the adult in the greater tendency to catarrhal symptoms always manifested. The attack comes on suddenly, generally without any warning, but occasionally preceded by a slight catarrh of the respiratory mucous membrane. Although it may develop at any hour of the twenty-four, it occurs oftener at night. It begins with wheezing respiration and dyspnea, this growing worse until there is orthopnea, the patient being obliged to sit up in bed with the head thrown back and the shoulders elevated in the effort to place the body in the most favorable position for breathing. There is vigorous action of the accessory respiratory muscles with inspiratory retraction and moving of the alæ nasi, as in cases of stenosis of the larynx. The mouth is open; the expression anxious, and the face pale, moist and cyanotic; the respiration very noisy, difficult and wheezing, with expiration much prolonged; the pulse rapid, feeble and small; there is no fever. The thorax exhibits little respiratory excursion, and auscultation shows very numerous loud wheezing râles of all sorts with feeble respiratory murmur. Percussion is tympanitic, with the cardiac dullness obliterated and the hepatic dullness diminished. As the paroxysm gradually subsides the râles in the chest grow coarser and there is some cough and more or less expectoration, the latter being clear or frothy, perhaps with numerous eosinophilic cells and possibly Charcot-Leyden crystals. Exhaustion and sleep follow. The attack lasts usually only a few hours but may be longer. It may be repeated daily or not for weeks or months. In the intermission there are no symptoms, unless the paroxysms are frequent and severe, in which event more or less complicating emphysema and bronchitis may be constantly present and the general health may suffer. In some of the cases dependent upon anaphylaxis to some certain protein the symptoms develop with the greatest suddenness after the ingestion of the food containing this, and may rapidly reach a most alarming intensity.

Hay Fever.—In this connection brief reference may be made to a periodical appearance of a form of bronchial asthma which is usually preceded in earlier years by characteristic autumnal coryza or rose-cold combined with conjunctivitis. It is rare in infancy and only in later childhood begins to show the asthmatic characters. Even then it usually still exhibits also the catarrhal condition of the upper respiratory passages. (See Hay Fever, p. 7.)

Complications.—A permanent emphysema with the resultant thoracic deformity may develop in severe asthma of either type. Always during the attack some degree of emphysema is present. Eczema and urticaria are liable to occur in many subjects between the attacks of asthma.

Prognosis.—That of *asthmatic bronchitis* in infancy and early childhood is good, as regards both life and ultimate recovery. The majority of cases cease to exhibit symptoms as the predisposition to bronchitis disappears with increasing age. So, too, when the asthmatic condition depends upon some removable cause, such as rickets or adenoid growths, the prognosis is favorable. If, however, it persists, and this in spite of treatment, there may be danger of it passing into bronchial asthma. The more frequent the attacks and the longer the duration of the disease, the more likelihood is there of a permanent emphysema remaining.

In *bronchial asthma* the prognosis is by no means so favorable. Death may take place during an attack, although this is of rare occurrence. The chance of the continued persistence of the disease is, however, decided, especially in those with a hereditary predisposition, or exhibiting a decided protein-sensitization; or in those cases which have lasted a considerable time in spite of treatment. Yet the chances of recovery are certainly greater than in adults, and in those in whom the attacks began quite early in life there is a tendency to amelioration or disappearance as maturity is reached.

Diagnosis.—In *asthmatic bronchitis* the diagnosis is not always easy, yet the nervous element which produces the asthmatic symptoms is not present in ordinary cases of bronchitis, while the dyspnea dependent upon disease of the heart or upon pneumonia is attended by other symptoms which will usually remove all diagnostic difficulty. A useful and very positive diagnostic sign of asthma of any form is the high eosinophilia, equalling from 10 per cent. to 20 per cent., which an examination of the blood reveals. Cutaneous tests with different proteins may show a susceptibility to some one or more of them. *Bronchial asthma* presents little difficulty of diagnosis. It can be confounded only with obstruction of the larynx or trachea from various causes which produce dyspnea; but in none of these is there the combination of prolonged expiration, expiratory dyspnea without involvement of inspiration, acute emphysema, and the numerous râles characteristic of asthma; while other symptoms are present not seen in the asthmatic disorder. The difficulty in diagnosis is less in the dyspnea from cardiac disease.

Treatment.—The most important treatment consists in a thorough search for the cause of the asthma and the removal of this if possible. Anemia or other evidence of impaired general health is to receive treatment; rickets should have that appropriate to it, with particular reference to the avoidance or cure of thoracic deformities; recurring attacks of bronchitis must be prevented by appropriate hygienic measures, and relieved as quickly as possible if they occur. (See pp. 6, 33.) The life should largely be an out-of-door one, and all mental overwork and excitement must be avoided. The confining of the child to the house between the attacks, on the ground that he takes cold if exposed, produces a deterioration of health and, in the long run, an increased susceptibility. Any obstruction or other disorder of the nasopharynx must be treated and the digestive functions should receive careful attention, the supper especially being made light and a careful study of the effect of each article being made, in view of the possibility of the existence of some anaphylactic protein-idiosyncrasy. In cases at all severe or troublesome moving to a different climate, especially in the winter season, may be an absolute necessity, and may prevent the disease becoming a permanent one. What climate shall be chosen is a matter of individuality, and this is particularly true of bronchial asthma. When the first climatic experiment is to be made, a warm and dry situation should be given the preference; but the seashore suits some well and the mountains others; while some are better in the fresh air of the country and others in the drier air of the city, particularly in autumn. High elevations are not suitable for subjects who have developed emphysema. Wherever it is found that the child is free from asthmatic attacks he should remain for a time sufficiently long to prevent recurrence; how long can be determined only by trial. If the climatic treatment is commenced early and persisted in thoroughly, entire recovery may often take place and a life of suffering be prevented.

In the way of therapy other than hygienic, tonic treatment is particularly indicated in *asthmatic bronchitis*, preferably cod-liver oil combined with such remedies as iodide of potassium, creosote, tar, terpin hydrate, and the like, as recommended for chronic bronchitis (see p. 38); including also inhalations of vapor containing creosote, eucalyptol or terebene. Counter-irritation applied to the chest is of service. With regard to the employment of drugs in *bronchial asthma*, the choice varies with the case; but on the whole treatment of this sort is unsatisfactory. Cod-liver oil, quinine, iron and arsenic find their place as general tonics. Iodide of potassium often appears to be of distinct benefit in the prevention of frequently occurring attacks and the mitigation of symptoms during their presence. Lobelia is an old-time remedy and has seemed to be of value, but should not be pushed to a degree to produce depression. Antipyrine given at bed-time sometimes seems to prevent the occurrence of a seizure. The dose should be from 5 to 8 grains (0.32 to 0.52) at 10 years of age. A number of remedies are in vogue for use during the attack. One of the most serviceable is the inhalation of the fumes from the burning of saltpeter, combined with stramonium leaves, given in a croup-tent or by some other measure which insures the inhalation of sufficient of it. Various asthmatic powders and cigarettes have been devised. A formula used at St. Bartholomew's Hospital[1] consists of powdered stramonium 1 oz. (31), powdered anise fruit ½ oz. (16), potassium nitrate ½ oz. (16), bruised tobacco 30 gr. (2). From 10 to 30 grains

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[1] Pharmacopeia of the London Hospitals, Squire, 1900.

(0.64 to 2) of the powder should be placed upon a plate and ignited. A smaller amount should be used at first in the case of children. A favorite remedy is the burning of blotting-paper which has been moistened with a 1:15 solution of nitrate of potash and dried. The inhalation of warm water-vapor is an excellent remedy. A dose of chloral will often relieve, given by mouth or by the rectum, and nitroglycerine or other nitrite is frequently of value; and one of the best of remedies is the hypodermic injection of morphine in small amounts; remembering, however, the danger from its frequent employment in cases where attacks are liable to be often repeated. An emetic is of value if the stomach has been overloaded. A hypodermic injection of a small amount of epinephrine (3 to 5 m. (0.19 to 0.31) of the 1:1000 solution) has often been of service. Atropine and many other remedies have been favorably recommended, and calcium salts are claimed by Göppert[1] and others to be capable of arresting an attack. Counter-irritation of the chest is an aid, and a general hot bath or a hot pack may relieve the spasm. The inhalation of oxygen can be tried if the dyspnea is great. Experiments have been made in the line of immunizing with minute, frequently repeated doses of a protein those subjects who have shown a protein-susceptibility. Success has sometimes been obtained; but, on the other hand, most alarming symptoms have been observed in some instances, and the procedure must be attempted with the greatest care.

BRONCHIECTASIS

(Bronchiolectasis)

Etiology.—This is a disorder probably more common in early life than is ordinarily supposed, although less frequent than in the adult. It may occasionally occur as a congenital condition, a portion of lung being replaced by saccular dilatations of a bronchial tube and its branches. Cases of this sort have been reported by Grawitz[2] and by Heubner;[3] and Peiser[4] was able to collect 31 published cases which he believed to have been of congenital origin. Carr,[5] Vogt[6] and others have published cases observed in the first 2 years of life. Most acute pulmonary diseases may be attended by a moderate dilatation of the bronchial tubes, which is temporary in nature and disappears with the recovery from the primary affection, owing to the natural elasticity and power of resistance of the bronchial tissues in early life. In other fatal instances of these diseases bronchiectasis may be found. It is only, however, the more chronic bronchiectasis which is now under consideration. Chronic bronchitis or bronchopneumonia, especially when developing after measles, grippe, and pertussis, may readily produce bronchiectasis of this more permanent nature; as may chronic interstitial pneumonia, and pleurisy with subsequent shrinking of the lung from overgrowth of connective tissue. (See Chronic Pneumonia, p. 90.) Foreign bodies in a bronchus, or any other cause of stenosis, may lead to bronchiectasis. In general a chronic bronchiectasis requires for its development a chronic pulmonary affection with more or less pulmonary sclerosis, the dilatation resulting from the shrinking of the lung.

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[1] Med. Klinik, 1914, X, 1003.
[2] Virchow's Arch., 1880, LXXXII, 217.
[3] Lehrbuch d. Kinderheilk., 1911, II, 286.
[4] Monatsschr. f. Kinderh., 1910, VIII, 602.
[5] The Practitioner, 1891, XLVI, 87.
[6] Jahrb. f. Kinderh., 1911, LXXIV, 627.

Pathological Anatomy.—The dilatations may be cylindrical, fusiform or sacculated, the latter being much the most frequent variety found at autopsy. They are multiple and oftener unilateral, and may occupy any portion of the lung, but are found most frequently in the lower lobe. The cylindrical and the fusiform bronchiectasis is open at each end, but the sacculated form opens into a bronchus, usually of middle size or small, and is closed in all other directions. Sometimes even this opening into the bronchial tube becomes closed and the dilatation then becomes a cyst. The mucous membrane of a bronchiectasis is atrophied and the cylindrical epithelium is replaced by that of the pavement variety or by granulation tissue. In advanced cases the other layers of the bronchial wall may be more or less destroyed. The size of bronchiectatic cavities ranges from less than that of a pea up even to that of a hen's egg or larger. The contents consist of a thin muco-pus with very numerous bacteria of different sorts, and possess an offensive odor. Pleural adhesions and sclerosis of the lung are generally found at autopsy.

Probably to be included under the lesions of bronchiectasis is the condition in which throughout extensive areas of the lung there are innumerable minute cavities, apparently dilatations of the bronchioles (*bronchiolectasis*), producing the appearance denominated "honey-comb lung."

Symptoms.—The disease generally comes on slowly and the time of its beginning cannot be accurately determined, although it may be suspected if the physical signs, especially the presence of coarse râles, are most marked in one locality. Fully developed persistent bronchiectasis requires usually several months for its evolution. There is then a paroxysmal loose cough, more marked in the morning or on change of position; productive in older children, with the expectoration of a somewhat large quantity of thin, muco-purulent secretion often having a very disagreeable odor and sometimes streaked with blood. Decided hemoptysis may take place, but is not common in the disease occurring in early life. Sometimes hours may pass without any cough whatever, or even several days before an emptying of the cavity occurs. The odor is not so offensive as in gangrene of the lung, yet some odor is nearly always present on the breath even when the expectoration is swallowed. The sputum when in large amount may on standing separate into the three layers often described as characteristic; an upper, thin and frothy; a middle, thin and clear or slightly turbid; and a lower, thick and purulent.

Examination of the lungs gives very variable results, and reveals decided physical signs only in cases of unusually large bronchiectatic cavities. Then numerous râles may occur on some occasions, and on others bronchial respiration and bronchophony or even amphoric respiration; while the percussion note may have a tympanitic quality, or at other times be impaired. Signs of cavity are, however, uncertain in character. The alteration in the physical signs from time to time depends upon whether or not the dilatations have been emptied of their contents. A moderate shortness of breath may occur after coughing or upon exercise, but decided dyspnea is not a usual symptom of the disease, and when present depends upon other causes in the lungs. Yet in some cases the patient may be obliged to sit upright all night or assume some other position because any except this is productive of an access of violent coughing. The general condition is good, the appetite not impaired, and there is no fever in the case of children able by the cough to keep the cavity well emptied. Otherwise fever, sometimes of a hectic type, may occur, and emaciation and loss of general health follow. In severe instances fever is seen occasionally, due to an intercurrent bronchitis, a small localized bronchopneumonia, or other complication. Decided clubbing of the fingers (*pulmonary osteoarthropathy*) is not uncommon in long-continued cases. This may, of course, occur in other diseases.

Complications.—The disease is oftener a complication or sequel of other disorders than itself the primary affection. These conditions have been mentioned in discussing its etiology. In addition intercurrent attacks of bronchitis or of bronchopneumonia can occur from time to time in cases of bronchiectasis, and may be the cause of symptoms. Pulmonary gangrene, pericarditis, peritonitis, or pleurisy, generally purulent, may develop, or metastatic abscesses in the liver, brain, spleen or elsewhere, or amyloid changes may result from the long-continued suppuration.

Course and Prognosis.—In acute temporary bronchiectasis the prognosis is good, the lesion disappearing with the primary disease. In the more permanent condition the course is very chronic, lasting perhaps for years, with little immediate danger to life except from the occurrence of complications. Eventually, however, the general health is liable to fail. The best prognosis in more persistent dilatation is in the cases dependent upon a foreign body in the bronchus. If this is expelled recovery may take place if the condition has not advanced too far.

Diagnosis.—This rests chiefly upon the periodic attacks of cough with profuse expectoration, and on the discovery upon examination of the lungs of localized coarse râles and sometimes of tympanitic percussion with bronchophony and bronchial respiration. When these signs are not well developed, difficulty may arise in distinguishing the disease from encapsulated empyema, and even exploratory puncture may mislead. Tuberculous cavities are attended by severe constitutional symptoms, and tubercle bacilli may be found in the sputum. Many cases at first thought to be tuberculosis are probably in reality instances of bronchiectasis. Chronic bronchitis with profuse expectoration may not be distinguishable from bronchiectasis unless physical signs of the latter can be found, although the paroxysmal discharge of a large amount of sputum at one time, with long intervals of freedom from coughing, is suggestive of bronchiectasis. The perforation of an empyema into a bronchus may at first suggest the emptying of a bronchiectatic cavity; but the much larger amount of pus evacuated on the first occasion and the much smaller amount later makes the diagnosis of empyema clear. Pulmonary gangrene runs a more acute course with severe constitutional symptoms, and the expectoration and the breath possess a gangrenous odor.

Treatment.—This can be symptomatic only. The general strength must be supported and increased by every possible means, above all by life in the open air or even by more or less permanent change of climate, as discussed in the treatment of chronic bronchitis. The inhalation of vapor containing oil of terpentine, oil of pine, eucalyptus, or creosote is useful in modifying the amount and character of the secretion. For the same purpose remedies of this class may be administered internally. Operation has been attempted in a number of cases. Soulegnac[1] has collected 45 instances of operation with 7 complete recoveries. The procedure is too dangerous to be recommended except possibly in instances of large localized bronchiectasis with increasingly severe general symptoms.

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[1] Ref., Lapin, Arch. f. Kinderh., 1903, XXXVII, 406.

CHAPTER V

DISEASES OF THE LUNGS (CONTINUED)

PNEUMONIA

(Inflammation of the Lungs)

Pneumonia is one of the most common and serious diseases of infancy and childhood, and in its various forms one of the most frequent causes of death. It is described under several forms, the two chief varieties being respectively (1) bronchopneumonia and (2) croupous pneumonia; and in addition to these reference must be made to (3) chronic interstitial pneumonia and (4) hypostatic pneumonia. The first two were first clearly differentiated from each other in the description by Rilliet and Barthez.[1] Although in typical cases the distinction is now firmly based on anatomical findings and clinical symptoms, there are very numerous instances in which the latter are far from being conclusive, and others which even at autopsy may be found to be hybrid forms, the two pathological processes being combined in the same portion of the lung, or one part showing a fibrinous inflammation, another the lesions of bronchopneumonia, and in some instances only a microscopical study revealing the true nature of the lesion. The anatomical differences consist in the existence of a fibrinous exudate into the vesicles, chiefly in a circumscribed form, in the case of croupous pneumonia, and a peribronchitis and peri-alveolitis in bronchopneumonia with exudation of cells into the alveoli, the process being more scattered and lobular in character. Yet a pneumonia which is fibrinous in its histological structure may sometimes have a lobular distribution; and, on the other hand, a bronchopneumonia may involve all or a large portion of one lobe and be confined to this. The titles "lobar" and lobular" are consequently misleading.

With this unavoidable uncertainty as to diagnosis so often present, statistics as to the relative frequeney of the two forms, based as they must be upon clinical symptoms in all but the fatal cases, are of but limited value, since the diagnoses are necessarily so often inaccurate, and fatal cases of croupous pneumonia in children are but few. It is these facts which explain the great variation in statistics published by different individuals and coming from different hospitals. Thus in 500 cases of pneumonia in children analyzed by Dunlop[2] there were in the first 2 years of life 45 of the croupous form and 233 bronchopneumonias, a ratio of 1:5.6; whereas in 370 cases analyzed by Holt and Howland[3] there were 80 of the croupous form in the first 2 years and 242 of the other, a ratio of 1:3 almost twice the ratio existing in the other series. Morse's[4] statistics are still further at variance, since of 178 cases of pneumonia in the first 2 years 118 were diagnosed as croupous and only 60 as bronchopneumonia, a ratio of 1.97:1. As the age increases croupous pneumonia becomes constantly more frequent and bronchopneumonia less so. The ratio in Dunlop's 500 cases for all periods of infancy and childhood is 1:2.4. As far as figures can be trusted, bronchopneumonia is, then, much the more common disorder during the first 2 years, and its mortality is certainly much greater than is that of the croupous form; while after the age of 4 years the latter is far in advance in the number of cases, and the former is of exceptional occurrence. My own opinion is, however, that of Comby,[1] and others that croupous pneumonia in infancy is probably much more frequent than many statistics would indicate.

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[1] Maladies des Enfants, 1838.
[2] Brit. Med. Jour., 1908, II, 367.
[3] Dis. of Infancy and Childhood, 1916, 495.
[4] Arch. of Ped., 1904, Sept.

Bronchopneumonia, being anatomically an inflammation of the bronchi as well as of the alveoli, is a link binding diseases of the bronchial tubes to those of the pulmonary tissue, and might properly be classified with the former. On the other hand, croupous pneumonia, as an undoubted infectious disorder, might be placed, as it sometimes is by writers, with others in that category. The consideration of the two together under the heading of diseases of the respiratory apparatus is a matter to be recommended on the ground that the study of the subject is simplified thereby.

ACUTE BRONCHOPNEUMONIA

(Catarrhal Pneumonia; Lobular Pneumonia; Capillary Bronchitis)

As a disease which involves in part the alveoli and in part the bronchial tissues, and in which the evidences of bronchitis are always present, this affection is very properly denominated "bronchopneumonia." The title "lobular" is to be avoided, since the disease may be distinctly lobar in type; and "catarrhal" does not fully express the pathological changes which occur. An inflammation of the smallest bronchioles without involvement of the alveoli, viz. "capillary bronchitis," is a condition which cannot be distinguished clinically from pneumonia, and its separate existence pathologically is open to doubt.

Etiology.—The influence of age is very decided; bronchopneumonia being preëminently a disease of infancy and early childhood, 75 per cent. or more of the cases occurring before the age of 2 years. After the 4th year it appears to be comparatively infrequent except as a complication in the course of various of the infectious fevers; until old age is reached when it is again observed. In 605 cases reported by Koplik[2] there were from 1 to 3 months 32; 3 to 6 months 68; 6 to 12 months 207; 1 to 2 years 298. Nothing is said of the incidence after this period. Holt and Howland[3] in 426 cases found in the 1st year 224; 2d year 142, 3d year 46, 4th year 10, 5th year 4. In 353 cases studied by Dunlop[4] 233 were in the first 2 years, leaving 120 for from 2 to 12 years of age. Sex appears to have but little influence, but hygienic conditions are factors of moment, crowding and other unsanitary conditions, as seen among the poor, increasing the susceptibility. The season of the year is a predisposing influence, much the greater number of cases occurring during the winter and spring. The existence of other diseases is of great importance. Consequently bronchopneumonia is conveniently divided into the *primary* and the *secondary* forms. In the former the disease develops independently of any previous condition of the health. It is especially in these instances that local chilling of the body appears often to be the sole active cause. In the decidedly more common secondary form, from 2 to 3 times as frequent, apart from the influence of chilling, which is often very evident, the disease occurs as a complication of diphtheria, pertussis, measles, grippe, typhoid fever, scarlet fever, sepsis, or other infectious disorder, the first four being the most frequent of these; or it develops in children debilitated by syphilis, rickets, or gastrointestinal affections. Bronchitis is a very common precursor of this secondary bronchopneumonia, and these cases might with propriety be called primary, since the pneumonia is then but a further development by extension of the bronchitic process. In severe cases of digestive disturbance bronchopneumonia is a very common terminal disorder. The majority of primary cases appear to occur in the first 2 years of life (92 out of 120, Dunlop); whereas half or more of the secondary cases are seen after this age (141 out of 233, Dunlop).

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[1] Traité des mal. de l'Enfance, Grancher, 1904, III, 399.
[2] Dis. of Infancy and Childhood, 1910, 632.
[3] Dis. of Inf. and Child., 1916, 493.
[4] Brit. Med. Journ., 1908, II, 367.

In early infancy some cases are caused by the entrance of food into the air passages after vomiting, or during efforts at swallowing, the so-called "deglutition" or "aspiration" pneumonia; and at later periods the accident may happen during unconscious or greatly debilitated states, or be the result of the entrance of a foreign body into a bronchus.

TABLE 83.—BACTERIA IN BRONCHOPNEUMONIA

(Eyre)

Microörganisms isolated	In pure culture	In association with one or more pathogenic bacteria
Diplococcus pneumoniæ	12	39
Streptococcus pyogenes longus	13	29
Staphylococcus pyogenes aureus and albus	4	25
Micrococcus catarrhalis	1	12
Micrococcus tetragenus	0	7
Bacillus of Friedländer	4	7
Bacillus influenzæ	3	15
Bacillus pertussis	0	2
Bacillus pyocyaneus	0	1
Bacillus typhosus	0	1
Bacillus diphtheriæ	0	3

The **exciting cause** is infection by a germ, but this may be of various sorts. The pneumococcus of Fränkel is probably that most frequently present in cases of primary bronchopneumonia. In secondary forms the streptococcus, staphylococcus, or pneumococcus, is perhaps oftenest seen; or the colon bacillus, micrococcus catarrhalis, diphtheria bacillus, pneumobacillus of Friedlander, influenza bacillus, or typhoid bacillus may occur. The tubercle bacillus is present in combination with other germs in the very frequent cases of tuberculous bronchopneumonia. In all forms of bronchopneumonia the infection is very often a mixed one, the germs being associated in a very varied manner, sometimes one species being most in evidence and sometimes another. Netter[1] in 42 autopsies on cases of bronchopneumonia in children obtained pure cultures in 25; these being pneumococcus 10 cases; streptococcus 8; staphylococcus 5; pneumobacillus 2. Including the cases of mixed infection the pneumococcus was present in 19; streptococcus in 23; staphylococcus in 12 and pneumobacillus in 6. Eyre[2] publishes the accompanying tabular analysis of the examination of 99 cases. Dürck[3] in 39 autopsies,

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[1] Arch. de méd. exper., 1892, IV, 28.
[2] Allbutt and Rolleston, Syst. of Med., 1909, V, 177.
[3] Deut. Arch. klin. Med., 1897, LVIII, 368.

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nearly all secondary bronchopneumonia, found the diplococcus pneumoniæ in 33; streptococcus pyogenes 14; staphylococcus pyogenes aureus and albus 21; bacillus pneumoniæ 12; diphtheria bacillus 11, bacillus coli 2; saprophytic bacteria 8. A monobacterial infection occurred in but 3 cases. On the other hand, Pearce[1] in 82 autopsies on bronchopneumonia in children after infectious diseases found the pneumococcus only 8 times.

As regards the influence of the pneumococcus considered as a group of organisms, the results of the investigations of Dochez and Gillespie,[2] Cole and Dochez,[3] and others, upon croupous pneumonia occurring in adults, have shown that the pneumococcus can appear in 4 different types, or more properly groups. Type I, the pneumococcus mucosus or streptococcus mucosus, and Type II are those most frequently found, and these, with Type III, produce the greatest mortality. The number of cases of Type IV equals about 25 per cent. and are of the mildest sort, and the death-rate is low. It is the last which is the group often found in the throats of healthy persons. These results as applied to children have been confirmed by Pisek and Pease,[4] Wollstein and Benson[5] and Mitchell,[6] but with certain important modifications; the conditions found obtaining regardless of whether the disease was bronchopneumonia or the croupous form. The fixed type of pneumococcus is much less often present in early life. Type IV is that most frequently found (Mitchell, 74.4 per cent., chiefly croupous pneumonia; Wollstein and Benson, 60 per cent., chiefly bronchopneumonia) and the mortality from it is greater than in adults. Type I is infrequently present.

It is a matter of discussion whether bronchopneumonia is contagious. The primary cases, at any rate, are certainly very infrequently so. In the secondary cases, especially those associated with measles, diphtheria, and other infectious disorders, a degree of contagiousness would appear to exist (p. 63). It is probable that the germ in most cases enters the bronchial tubules from the oral and the upper respiratory mucous membranes. The germs in the mouth and nasopharynx remain harmless until some of the predisposing agencies remove the resisting power of the mucous membrane of the bronchi; after which the penetration of this by the microörganisms with consequent inflammation becomes possible. In the case of bronchopneumonia following gastrointestinal disorders, infection by way of the blood seems possible.

Pathological Anatomy.—The basic lesion in bronchopneumonia is an inflammation of the walls of the bronchi and of the air vesicles about them. A small-celled infiltration takes place into the walls of the smaller bronchi and the peri-bronchial connective tissue, and extends thence not only to the terminal bronchioles but also to both the terminal and surrounding air-vesicles and the interacinous pulmonary tissue. The walls of the bronchi and alveoli are decidedly thickened and greyish in color, and the lumen filled with an exudate consisting of dead epithelial cells, leucocytes, and often fibrin, the latter in small amount and not to the extent seen in croupous pneumonia. The neighboring capillaries are engorged. In some of the acute primary cases the lesions do not so much appear to spread from the bronchi to the alveoli as to develop simultaneously in both. The pathological process produces scattered foci, at first small with the thickened bronchioles as the centre, and from these a droplet of pus can be squeezed. The confluence of neighboring pneumonic foci results in larger areas of varying size, rather firm and projecting on section, and in color at first red and later of a greyish or yellowish tint. The smallest are little larger than a miliary tubercle. The portions of uninvolved pulmonary tissue between the consolidated areas are soft, edematous, congested, and often atelectatic, sometimes emphysematous. There is thus brought about a decided contrast between the hard, prominent, greyish-red pneumonic foci and the purplish red, edematous, congested or atelectatic regions. There may be observed, too, a contrast of color between neighboring lesions dependent on the respective differences in their age, the fresher ones being still red, the older ones grey. All this gives the lung a somewhat mottled appearance. The conditions are the reverse of those seen in croupous pneumonia, the affected areas in that disease being of a deeper red color than the healthy lung.

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[1] Bost. Med. and Surg. Jour., 1897, CXXXVII, 561.
[2] Journ. Amer. Med. Assoc., 1913, LXI, 727.
[3] Trans. Assoc. Amer. Phys., 1913, XXVIII, 606.
[4] Amer. Jour. Med. Sci., 1916, CII, 14.
[5] Amer. Jour. Dis. Child., 1916, XII, 254.
[6] Journ. Penna. State Med. Assoc., 1917, XX, 343.

Reference has been made to the presence of atelectatic and of emphysematous areas. The former are produced by occlusion of the bronchioles by secretion, with consequent collapse of the pulmonary tissue supplied by them. It is a disputed question whether the development later of inflammation in these vesicles is a result of this collapse. The atelectatic areas are found especially in the neighborhood of the pneumonic foci. The emphysema depends largely upon the compensatory distention of vesicles and the mechanical effect of coughing. It is scattered among the consolidated areas, but occurs principally on the anterior edges of the lungs. The rapidity, method, and degree of extension of the pneumonic lesions is very variable. In some cases there is a rapid, diffuse development of the process with death following in a day or two. In these, large areas of the lung, especially the posterior portion, exhibit apparently no consolidation, but only intense uniform congestion and edema, with the bronchioles filled with pus. The anterior parts are pale and emphysematous. Microscopical examination, however, shows the congested lung to be in the earliest stages of the bronchopneumonic process. In cases lasting several days up to 2 weeks the mottled appearance described is found. The disease may have spread very slowly and not to any great extent in cases of comparatively short duration; but in those lasting longer it gradually invades much of the lung and most frequently both lungs, and the bronchopneumonic foci are widely diffused. As a rule the external portion of the lung is that chiefly involved and most of the consolidated areas are visible from the surface; others more centrally situated are discoverable only on section of it. In many cases the extension has been chiefly in one region, and the confluence of the foci has finally produced a large consolidated area, sometimes occupying the greater part of a lobe. To this the title "pseudolobar bronchopneumonia" is applied. In cases much prolonged, the section is largely grey in color and minute or larger abscess-cavities may be found.

With regard to the situation of the lesion, the posterior portion of the lungs are the parts oftenest involved. In the greater number of cases both lungs are attacked. The distribution in Dunlop's[1] 353 cases was: Both lungs 182; right lung alone 83; left lung alone 72; not stated 16. The bases were attacked 5 or 6 times as often as the apices.

In addition to the changes in the lung pleurisy is a very constant attendant, developing over the surface of the consolidated areas if of large

[1] *Loc. cit.*

size, and producing adhesions between the pulmonary and parietal pleuræ in these parts. The firmness of these adhesions depends upon the duration of the disease. A small amount of serum may be present in the pleural cavity and occasionally the effusion may be purulent. Enlargement of the bronchial glands is always found. Gangrene of the lung is an uncommon condition. Resolution in bronchopneumonia does not take place in the manner seen in the croupous form, in which the fibrin is rapidly absorbed from all parts of the affected tissue; nor do the lesions necessarily exhibit the different stages of congestion, hepatization and resolution. In bronchopneumonia the gradual spread of the disease is followed by gradually progressing absorption, and different parts of the lung show different stages as compared with each other. In cases which have been much protracted, overgrowth of the connective tissue in the alveolar septa, walls of the bronchial tubes, and pleura may take place to such an extent that complete resolution cannot occur and chronic interstitial pneumonia results. (See Chronic Pneumonia, p. 90.)

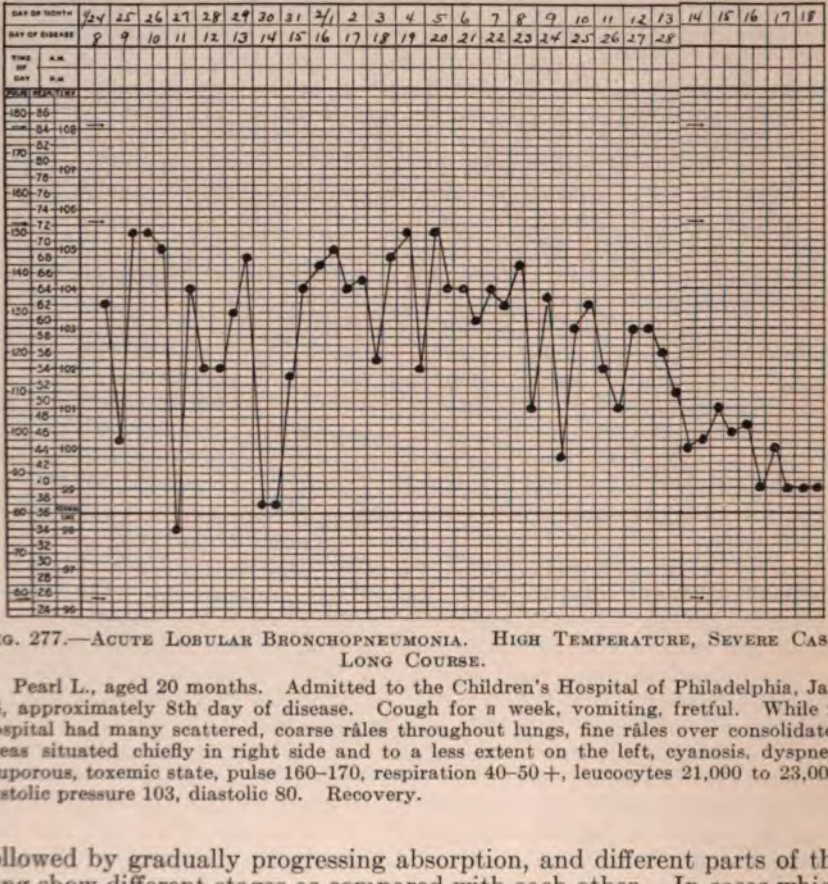

FIG. 277.—ACUTE LOBULAR BRONCHOPNEUMONIA. HIGH TEMPERATURE, SEVERE CASE, LONG COURSE.

Pearl L., aged 20 months. Admitted to the Children's Hospital of Philadelphia, Jan. 24, approximately 8th day of disease. Cough for a week, vomiting, fretful. While in hospital had many scattered, coarse râles throughout lungs, fine râles over consolidated areas situated chiefly in right side and to a less extent on the left, cyanosis, dyspnea, stuporous, toxemic state, pulse 160–170, respiration 40–50 +, leucocytes 21,000 to 23,000, systolic pressure 103, diastolic 80. Recovery.

Symptoms.—No very exact classification of the varieties of bronchopneumonia is possible, since the clinical symptoms and anatomical lesions are not always in accord. A number of different divisions have been made by writers. As factors influencing the clinical type are the age of he patient, the existence of other diseases, the duration of the attack of pneumonia and the nature and degree of the symptoms and physical signs. Although no one form can be called the type, the first of the following varieties is that most frequently seen and may be called the ordinary one.

1. Acute Lobular Bronchopneumonia.—In well-marked cases the symptoms begin acutely, perhaps after some days of preceding nasal and pharyngeal catarrh or of tracheo-bronchitis. There is a sudden rise of temperature accompanied often by vomiting and sometimes ushered in by a convulsion, although this is less often seen than in croupous pneumonia. In cases where bronchitis was already present, the severity of the symptoms may increase more or less gradually, with or without initial vomiting, the cough becoming dryer and more suppressed and painful, respiration more rapid and labored, and the temperature higher. In any well-developed case there is rapid dyspneic respiration, prostration, more or less cough, and cyanosis. The pulse is rapid, the appetite lost, there is restlessness or apathy, and the child looks ill.

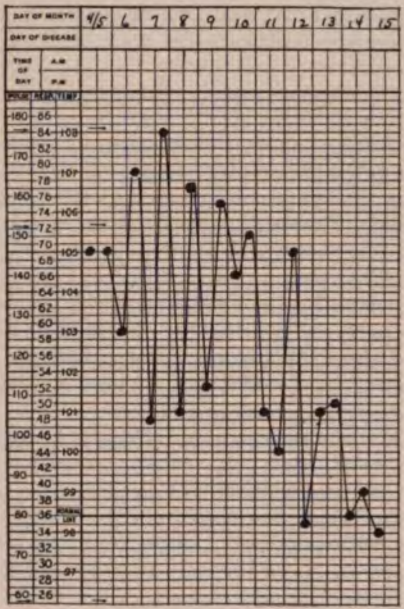

Fig. 278.—Acute Lobular Bronchopneumonia with Very High and Irregular Temperature.

Muriel D., aged 2½ years. Chart begins approximately on the 5th day. Gradually increasing consolidation of both lungs, principally of left apex and right base. Some degree of delirium, cyanosis and cardiac weakness. Respiration only moderately accelerated. Short course. Recovery.

The individual symptoms must be described more in detail.

The **temperature** may rise rapidly to 104 or 106°F. (40° or 41.1°C.) and is in many cases continuously high. As a rule, however, there is a more gradual rise with a tendency to remittance not seen in croupous pneumonia, often with daily drops of from 3° to 4°F. (1.6° to 2.2°C.) (Figs. 277 and 278). The degree of elevation of temperature is no necessary indication of the severity of the disease. Although in the mildest cases it is more likely to be lower; some of the more severe ones may never exhibit a temperature of over 101 to 102°F. (38.3° to 38.9°C.). Sometimes a terminal hyperpyrexia of 107°F. (41.7°C.) or over occurs just before death; but I have seen this high temperature in cases which recovered (Fig. 278). In much debilitated subjects especially in infancy the temperature may occasionally remain afebrile, or even subnormal throughout. The fall of temperature is nearly always by lysis. Not infrequently there is a fall followed by another rise; often indicating the spread of the pneumonic process to other parts of the lungs.

The **respiration** is always accelerated and the lack of proportion between the rate of the respiration, and that of the pulse is often striking, the ratio reaching sometimes 1:2 instead of the normal 1:3 or 4. The rate is generally over 50 and one of 70 or 80 per minute is not uncommon, and it may sometimes decidedly exceed this. I have seen it in one instance reach 150.

Dyspnea is a constant symptom. In the mildest cases it may be insignificant and shown only by slight moving of the alæ nasi when the child is under some excitement or making some exertion. Moving of the alæ cannot alone be taken as a certain sign of pneumonia, since it may occur even under the mere influence of nervous excitement. In well-marked cases, however, the alæ nasi dilate with each inspiration; there is episternal and epigastric retraction, inspiratory retraction of the sternocleidomastoid muscles and of the attachment of the diaphragm, and sinking in of the costal interspaces. A moaning or grunting cry—the "expiratory moan"—may accompany many of the expirations, but loud crying is uncommon, owing to the difficulty in getting breath. Not infrequently the respiratory rhythm is altered, the pause coming after inspiration instead of expiration as in normal breathing.

Cough is a very variable symptom. Often it is dry, painful, harassing and causing much loss of rest. In other cases there may be so little cough that it is almost overlooked. This may occur in either mild or severe cases and in the latter is an unfavorable symptom, indicating that the patient has not sufficient power to relieve the bronchioles of secretion. There is no expectoration in infancy and early childhood; the secretion being swallowed or drawn back into the lung.

The **circulatory system** exhibits always accelerated pulse, but in average cases generally of good strength and regular. In bad cases it grows weak and thread-like. The tension is usually lowered, except in the beginning, but for this there appears to be no certain rule. The pulse-rate may reach even 200 per minute in young subjects with severe attacks of the disease. Cyanosis is present to some extent in the well-marked cases. This may show itself only in a slightly bluish tint of the lips and fingers and of the flush of the cheeks. In a severe attack the whole surface of the body may be pale and bluish. The sudden development or increase of cyanosis may indicate a congestion or collapse of some portion of the lung previously unaffected. It may be attended by a sudden increase of dyspnea.

Prostration is observed to some extent in all cases, and in serious ones may be very great and be attended by much loss of weight.

Digestive symptoms are common in infants and of great importance. Tympanites may become great, and is of serious import in those quite ill with bronchopneumonia, inasmuch as it adds to the difficulty in thing. It is seen oftenest in rachitic subjects. Vomiting frequently rs in the disease and is not uncommon later. It may occur only sionally, or repeatedly and be resistant to treatment. Diarrhea is sometimes very troublesome, being most frequently encountered and most dangerous in summer time. Both vomiting and diarrhea usually depend upon the debilitated digestive power attending the pneumonia, although in some cases a true enteritis develops. Both are oftenest met with in the first 2 years of life. The appetite is usually greatly diminished and even when present the dyspnea may interfere with the taking of food.

Nervous symptoms are common especially as the disease advances. Convulsions may usher in the attack but not so frequently as in croupous pneumonia. Occurring later they are of serious import. In some cases there is great restlessness particularly if the respiratory symptoms are severe, when jactitation from air-hunger may develop. Delirium may occur, or the patient be unusually excitable. Oftener, however, there is fretfulness and sleeplessness; or a decided degree of apathy and even a stuporous state; and sometimes complete unconsciousness. In all well-developed cases the children look ill and frequently lie with half-open eyes.

The **blood** exhibits early a leucocytosis especially of the polymorphonuclear cells. The number of leucocytes usually equals 20,000 or decidedly more per cubic millimeter. Heiman[1] found an average of 33,900 in 19 cases in infancy. The **urine** shows only the ordinary changes characteristic of febrile diseases.

Physical Signs.—The physical signs are very variable even in cases of this more common type. Generally they develop somewhat slowly and may be discovered only after several days of illness and often, indeed, not until the case is well advanced. The first signs are usually the presence of fine râles, combined frequently with coarser ones, limited to some small area or areas, or most marked there. These areas are oftenest in the posterior part of the lower lobe or in the intervertebral spaces. They may be too small to produce any alterations of the percussion note. Later the râles become more widespread and change of the respiratory murmur is present, but this may be of a degree only sufficient to be classed as bronchovesicular and is characterized chiefly by a harsher expiration heard if the patient breathes deeply, the respiration at other times being often feebler than normal. Increased vocal resonance also is now audible, although perhaps only at the time of a cry, or consisting in the more clearly ringing character of the râles at the affected spot and in their nearness to the ear. There may by this time be very slight dullness elicited only by gentle percussion. In many cases, however, the voice-sounds are feeble and respiration so superficial that no positive auscultatory change can be discovered, and a slight dullness on percussion may be the only suggestive sign. Sometimes the percussion note over the affected region is at first fuller and more hyperresonant than normal owing to the presence of neighboring areas with distended vesicles. Very often in bronchopneumonia the emphysematous free border of the lungs in front diminishes the normal hepatic and cardiac percussion-dullness. Vocal fremitus generally gives unsatisfactory signs owing to the high-pitched and less resonant voice of the child. As the case advances no further change in the physical signs may occur; or in other instances there slowly develops decided consolidation over larger areas, and the usual loud bronchial respiration, bronchophony, and dullness on percussion observed in the croupous pneumonia of adults is discoverable. Always, however, there is a large admixture of coarse and fine râles, and friction sounds may sometimes be heard. In some instances physical

[1] Arch. of Ped., 1905, Oct.

signs suggesting cavities are present, whether or not these actually exist. There is seldom the sharply outlined demarcation of the consolidated area observed in croupous pneumonia, but this area blends gradually with the neighboring healthy tissue, and râles may perhaps be heard everywhere. The pneumonic process is seldom limited to only one region in typical cases; but eventually scattered areas of the disease are usually present in both lungs, especially in the lower lobes.

Always bronchopneumonia is characterized, at least early in the disease, by the lack of correspondence between the severity of the symptoms and the degree of the development of the physical signs. In very many cases percussion dullness remains absent throughout or until late in the attack, and auscultatory signs are often not discovered at all, or only if the child is made to cry, and the diagnosis must consequently rest upon the symptoms and not the physical signs.

2. ACUTE TOXEMIC BRONCHOPNEUMONIA. (*Acute Congestive Form*).—In some extremely severe cases of bronchopneumonia, especially as seen at times in early infancy, and either primary or secondary in nature, the process may not go beyond the stage of widespread intense congestion of the lungs, and the general symptoms appear to be dependent upon the toxemic condition. There is hyperpyrexia, great prostration, cyanosis, rapid respiration, and nervous symptoms consisting of delirium, stupor or terminal convulsions. Râles may be entirely absent and no physical signs of any sort discoverable which enable a diagnosis to be made. There may be a painful, frequent cough, or none at all. The symptoms are very much like those of some malignant cases of any of the acute infectious fevers. The course is usually short and death may occur in 1 or 2 days. In other instances the intensity of the symptoms diminishes and recovery is rapid; or the case may assume the symptoms and run the ordinary course of bronchopneumonia as usually seen.

3. ACUTE DISSEMINATED BRONCHOPNEUMONIA.—There occurs in this form, which is oftenest seen in infancy, a widespread involvement of the finer bronchioles throughout much of both lungs, with filling of these with secretion. Although the alveoli are also involved, the symptoms are those principally of a severe suffocative bronchitis. The title "capillary bronchitis" has been applied to this variety. After a short period of mild respiratory catarrh, there is a rapid development of high fever, accelerated respiration, extreme dyspnea, decided cyanosis and severe cough. Numerous râles of all sorts are widespread throughout the chest, but there are no signs of actual consolidation. The percussion note may even be hyperresonant from the presence of emphysema. Life is threatened apparently especially by the suffocative condition.

4. PSEUDOLOBAR FORM OF BRONCHOPNEUMONIA.—This differs little in symptoms from the form first described, except that there is often greater tendency for the temperature to remain steadily high. The physical signs are however, unlike. The pathological process is confined, at least at first, to one region of lung, and the rapid union of adjacent foci of infiltration soon produces an area of consolidation which occupies the greater part of one lobe and gives physical signs almost exactly like those seen in croupous pneumonia. There are fewer râles than in the ordinary type of bronchopneumonia. The disease is sometimes recognized by the fact that the pneumonic areas were at first distinct from one another, and that later in the attack evidences of consolidation are liable to be found in other parts of the chest. Yet I have seen the pseudolobar type

begin so suddenly, and so rapidly produce pseudolobar consolidation, that the diagnosis from the croupous form was impossible (Fig. 279).

5. Latent Form of Bronchopneumonia.—This is seen in very young infants, in older ones suffering from severe gastroenteric diseases, and in still older children unconscious and much debilitated in the course of some serious disorder. The onset is very insidious. Fever due to the pneumonia may be thought to be dependent on the primary condition, or there may be no fever whatever, no cough or dyspnea, and nothing in fact to draw attention to the lungs. As a rule, the disease is discovered only by accident or not until autopsy.

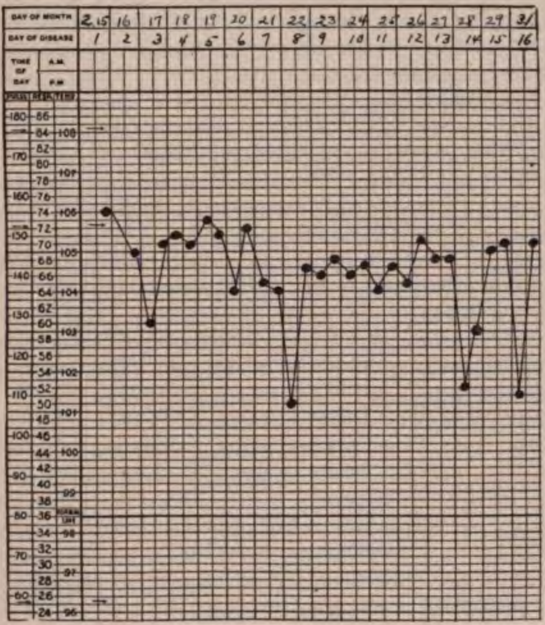

Fig. 279.—Primary Bronchopneumonia, Pseudo-lobar Type.

Andrew R., aged 2½ years. Sudden onset with convulsions on Feb. 15. On next day physical signs of consolidation in the right lung, extending thence over the entire upper part of the right lung in lobar form. No physical signs in left lung. Restlessness, delirium, at times opisthotonos, persistent high temperature with few remissions, increasing prostration, cyanosis. Many attacks of profound collapse. Death Mar. 1. Autopsy and microscopical examination showed pseudo-lobar bronchopneumonia of right lung, left lung free.

6. Protracted Form of Bronchopneumonia.—The protracted form is to be distinguished from the chronic interstitial form presently to be described in that the last is a sequel of pneumonia, the other an active process with lesions and symptoms of a different nature. It is seen principally in weakly infants or children, or occurring secondarily to the infectious diseases, especially pertussis, diphtheria and measles. It at first resembles the ordinary lobular or the pseudolobar form in symptoms and physical signs; but the former fail to ameliorate, except that the height of the temperature is often less, and the latter increase in extent and in degree of development while those suggestive of cavity appear; all this at a time when ordinarily improvement would be expected. There may sometimes be temporary remissions in the severity

of the symptoms, but, on the whole, loss of strength and weight is progressive until an extremely cachectic state is reached.

7. Aspiration Pneumonia.—This variety, called also "deglutition pneumonia," is distinguished rather by the cause than by the symptoms. It occurs in conditions of great cachexia, in coma or stupor, or after tracheotomy or intubation, and is the result of the entrance into the lung of food, or, in the case of the new born, the aspiration of amniotic liquid (Silbermann).[1] The exudate in the lungs in these cases is more purulent than in other forms. If the accident does not produce abscess or gangrene, there results a bronchopneumonia of the lobular type.

8. Tuberculous Bronchopneumonia.—The very common form of pneumonia associated with the tubercle bacillus may occur as a primary disease or be secondary to other disorders, especially measles and pertussis, or to tuberculous processes elsewhere in the body. The pathological lesions and clinical symptoms have already been described in discussing Tuberculosis. (See Vol. I, pp. 550, 553.) It may be pseudolobar but is oftener lobular in type. The symptoms are often practically identical with those of non-tuberculous bronchopneumonia; but in other cases the process develops more gradually, the symptoms are less acute and the course slower. As a rule, in cases of the first kind diagnosis is impossible except by examining the sputum for tubercle bacilli; although the fact that the lesions when localized are oftener found in the upper lobe is very suggestive. When bronchopneumonia develops in subjects clearly tuberculous, or when, in the course of a pneumonic affection, other symptoms suggesting tuberculosis appear, such as those of meningitis, the diagnosis of a tuberculous pulmonary lesion is rendered probable.

9. Bronchopneumonia Secondary to Other Diseases.—No reference is intended here to the occurrence of pneumonia following bronchitis, as that has already been described. In many secondary cases especially after infectious diseases, the symptoms of these are altered by, or modify those of the pneumonia. With *measles*, pneumonia may develop at any time in the course of the disease, but is oftenest seen in the stage of eruption or as it fades. During the eruptive period the temperature remains elevated and the symptoms and signs of pulmonary consolidation develop, the cough becomes more troublesome, dyspnea appears and localized fine râles may be added to the coarser râles previously present. Any of the types described may occur, but the disseminated and the protracted forms are frequent, or the pseudolobar may be seen with close resemblance to croupous pneumonia. The process may be simple or tuberculous. Occasionally the pneumonia appears with the symptoms of invasion in the infectious diseases and is then often rapidly fatal. In *pertussis* bronchopneumonia is prone to make its appearance after the disease has reached its height or is in the stage of decline. The onset is oftenest insidious; the symptoms and physical signs of the bronchitis present having gradually added to them those of pneumonia, with rise of temperature which generally does not become high, marked dyspnea, cyanosis, and the evidences of a severe pneumonic process. The paroxysmal cough of pertussis may be to a large extent replaced by the non-paroxysmal cough of pneumonia. In other instances the onset is much more abrupt; but in any event there is the change from comparative health between the paroxysms to a condition of decided illness. The disease is oftenest of the disseminated or the protracted type, or very frequently tuberculous in nature.

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[1] Deut. Arch. klin. Med., 1884, XXXIV, 334.

In *scarlet fever*, the development of bronchopneumonia is much less frequent than in measles and pertussis. Its onset is usually evident except in the exhausted septic cases, where it may be readily overlooked, since the fever would be assigned to the original disease. This form of pneumonia is very often attended by purulent pleural effusion. As a complication of *grippe* pneumonia is common and may be either of the ordinary or of the pseudolobar form. The onset may be sudden and the existence of grippe entirely overlooked, or the process may develop during the height of or as a sequel to the primary disease. In the epidemic of grippe of 1918 the cases of pneumonia were often very severe, and many of them exhibited a peculiar tendency to an absence of leucocytosis. (See Vol. I, p. 479, Fig. 151.) In other cases there was a failure of the ordinary increase of the rate of respiration. The tendency to the development of empyema as a sequel was also noteworthy.

Diphtheria of the larynx not infrequently has bronchopneumonia as a complication. In other cases of diphtheria the pneumonic process develops as a secondary result of the septic condition. Where intubation has been performed, the diagnosis is difficult since the presence of the tube alters the character of the auscultatory signs.

The complication of *gastroenteric disease* by pneumonia has already been referred to in considering the latent form of the latter disorder. The pulmonary involvement is often a terminal process, and the absence or slight development of fever with the greatly debilitated condition of the infant renders the pneumonia often entirely unsuspected.

Rachitis, when severe, is liable to be complicated by a bronchopneumonia of a very protracted form, and sometimes with little fever. The deformity and lack of firmness of the thoracic walls make respiration rapid and very dyspneic, the symptoms being increased by the special tendency to atelectasis present.

Complications and Sequels.—Of these pleurisy is the most frequent. A pleurisy attends the majority of cases in which the lesions are quite extensive. It was found present in 18 per cent. of 418 autopsies on bronchopneumonia in nurslings, reported by Reano.[1] It is oftener dry than serous. As recognizable from a clinical standpoint it is not as common a complication as in croupous pneumonia. In some instances empyema may follow, but this, too, is less frequent than in the croupous form of the disease. Purulent otitis media is very common. Tuberculosis may develop as a sequel; but most cases exhibiting lesions of this nature probably suffered at the beginning with a broncho pneumonia of the tuberculous variety. Diarrhea is common in the summer season. Bronchiectasis and emphysema may persist as sequels in long-continued cases. Gangrene of the lungs and pneumothorax are unusual. Various purulent inflammations of the skin may develop after severe protracted cases. Nephritis, purulent arthritis and osteomyelitis are only occasionally seen. Meningitic symptoms are not infrequently observed and a serous meningitis may occur; but one of a purulent nature is not frequent. Pericarditis, perhaps purulent, is a rare complication.

Relapse and Recurrence.—Increases of the elevation of temperature accompanying fresh extensions of the pathological processes in the lung are almost a part of the course of bronchopneumonia. True relapses are also common. These consist in a return of fever and of other symptoms and physical signs of the disease from a few days to a week, or sometimes more, after convalescence had apparently begun.

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[1] La Pediatria, 1913, XXI, 588.

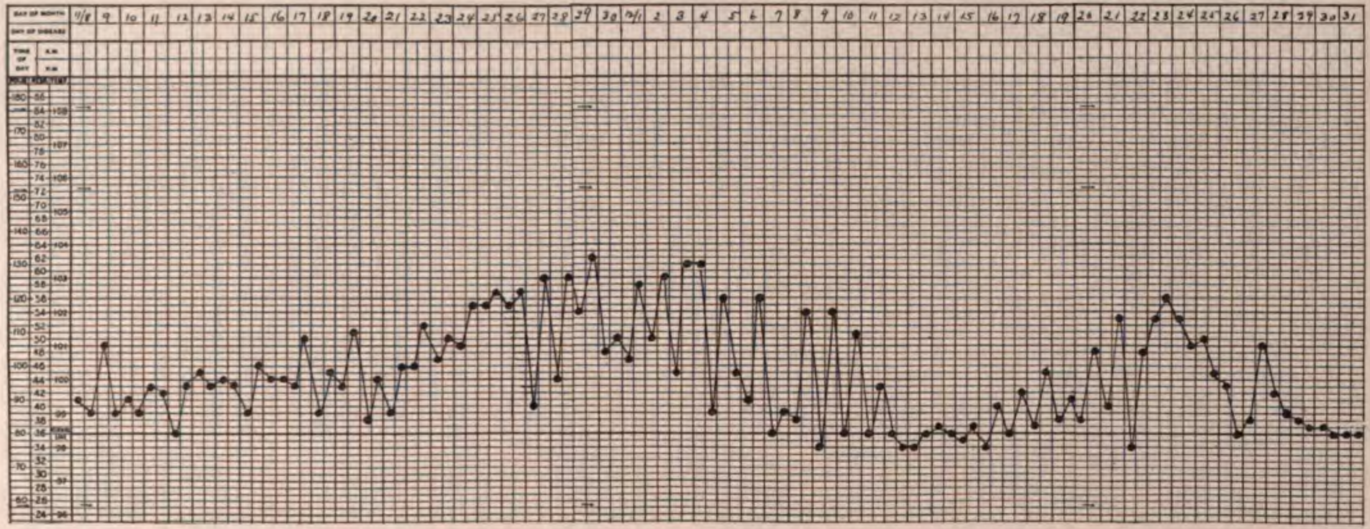

Fig. 280.—Acute Lobular Bronchopneumonia. Long-continued and Very Severe Course, Relapse, Delayed Resolution.

Helen G., aged 10 months. Patient in the Children's Hospital of Philadelphia. Slight cough began about Nov. 1. Brought to hospital Nov. 8, on account of illness in family, child well; Nov. 14, a few scattered râles in chest; Nov. 15, slow rise of temperature began; later developed extremely severe, persistent cough, relieved only by croup-tent; gavage needed on account of anorexia; dyspnea, cyanosis, rapid pulse and respiration, great prostration, death seeming imminent at times, very numerous râles, slowly increasing consolidation, with final involvement of greater part of both lungs; Dec. 11, apyretic; Dec. 14, lungs clearing, but still consolidated at back; Dec. 21, relapse began, with involvement of upper part of left lung which had cleared; Jan. 6, consolidation still present at right base, child convalescent.

(See Fig. 280.) This may take place in another part of the lung previously unaffected, or in one in which resolution has been practically completed.

Recurrence, in the sense of the reappearance of the disease in children entirely well, is not infrequent. Indeed, the fact that the child has previously had bronchopneumonia appears to a certain degree to be a predisposing factor favoring the development of another attack.

Relapse or recurrence may be more or less severe than the first attack. In 1 instance in a child of 8 months a second development of the disease was seen 4 or 5 weeks after the first, and a third and fatal attack 13 days after the second. Repeated relapse, as seen in the protracted form, may lead to the development of a chronic bronchopneumonia.

Course and Prognosis.—These vary greatly with the type of the disease. The duration of the average case of *lobular bronchopneumonia* is 2 to 3 weeks, the milder cases lasting often decidedly less than this, and many severe ones considerably longer (Fig. 280). The temperature continues sometimes high, but oftener irregular throughout, with falls almost to normal, there being no characteristic curve. With each extension of the process in the lungs, there is a fresh increase of fever. The symptoms of respiratory involvement continue marked and increase as the process extends through the lung, and prostration becomes very decided as the disease progresses. As it approaches a favorable termination the general symptoms improve and the temperature gradually falls, although it is still liable to sudden unexplained elevations. In fatal cases there is no improvement of the symptoms, the dyspnea and rapidity of respiration become very great, the pulse grows weak and increases in rapidity, cyanosis becomes marked and there is a steady progression toward a fatal ending, oftenest from respiratory embarrassment. *Acute toxemic* cases usually die in from 1 to 2 days.

In the *disseminated* form the course is short, the fever is usually moderate, accelerated respiration and dyspnea appear very early and continue, the cough is labored and severe, and cyanosis develops promptly and is marked. The symptoms rapidly increase in severity in fatal cases and the temperature often becomes hyperpyrexial, before death from asphyxia or heart-failure occurs on the 3d or 4th day. In patients recovering the symptoms may rapidly ameliorate or may change into those of the ordinary type.

The *pseudolobar form* does not differ from the ordinary type in its course except that the temperature is sometimes more regular. In the *latent form* the symptoms remain uncharacteristic throughout, cough may be absent, and fever absent or slight. The duration of the disease is uncertain since its onset is usually not discovered. The prognosis is extremely unfavorable.

In the *protracted form* prostration steadily increases, a cachetic state develops, fever persists but is not high and is very irregular, and death takes place after 5 or 10 weeks; or final recovery occurs, the symptoms and physical signs changing slowly, and the evidences of general ill health being still slower in disappearing. The prognosis is, on the whole, unfavorable, more cases dying than recovering.

The *prognosis in general* in bronchopneumonia is always doubtful. The difficulty in making an accurate diagnosis in many cases, and the personal element of the investigator which consequently exists, render statistics variable and misleading. Figures of the death-rate range all the way from 25 to 75 per cent. It might be safely placed at from 10 to

25 per cent. in private practice, and much higher in institutions. It is influenced by many conditions. The primary cases are the most favorable, the death-rate in bronchopneumonia secondary to other diseases being largely increased. When complicating pertussis the mortality is very high (Zienessen,[1] over 50 per cent.). In bronchopneumonia attending measles the mortality is also great (Comby,[2] 81.39 per cent. in 85 cases). In that developing during diphtheria nearly all die. Rachitis and gastroenteric disorders likewise influence the prognosis unfavorably. Age exercises a noteworthy effect on the mortality. In cases due to the pneumococcus, especially when in pure culture, the course seems generally shorter and the prognosis better than in other forms. (See also p. 50 on influence of different types of pneumococcus.) In general the younger the patient the more liable he is to succumb. The mortality percentage is consequently decidedly highest in the first 2 years of life, and in the 1st year half or more of the cases die. Unfavorable hygienic conditions of any sort increase the danger of the disease and the mortality percentage.

Various *individual symptoms* have a prognostic significance. Persistent hyperpyrexia of over 105°F. (40.5°C.) is a bad symptom. There is no unfavorable significance in a brief development of high temperature. It is the general trend of the pyrexia which is important. On the other hand, the low temperature of the latent form is not an auspicious indication. Among other unfavorable signs are early appearance of cyanosis or other evidences of cardiac weakness; marked pallor of the skin; great dyspnea and decided irregularity of the respiration; extensive involvement of the lungs; persistent excessive tympanites, disappearance of cough in patients not otherwise improving, great prostration and emaciation, persistent diarrhea; prolongation of the attack for over 4 weeks; repeated convulsions during the attack; and evidence of tuberculosis elsewhere in the body.

Diagnosis.—The diagnosis of bronchopneumonia from the *croupous* form is often one of great difficulty. This will be considered later in discussing croupous pneumonia. Here may only be said that the symptoms and physical signs in bronchopneumonia are in general slower in appearing, grow more gradually worse, last longer, and exhibit a severity much greater than in croupous pneumonia. From *bronchitis* bronchopneumonia of the ordinary type is to be distinguished by the higher elevation of temperature, greater evidence of illness, increased rapidity of respiration, decided dyspnea, presence of leucocytosis, and, later, the development of some of the localizing physical signs described. Severe bronchitis however, cannot always be distinguished from bronchopneumonia. There may in it be decided dyspnea and the fever may be high. This fever, however, soon falls. There is a wider diffusion of râles, which are coarse rather than fine. Localized râles and persistence of fever after 3 or 4 days indicate the probable existence of bronchopneumonia. Acute bronchopneumonia is frequently preceded by a bronchitis. The transition is characterized by gradual increase of temperature and of rapidity of respiration, with the development of dyspnea and of decided prostration. Acute disseminated bronchopneumonia especially cannot always be differentiated from severe bronchitis, but in the former the fever is usually higher and finally often hyperpyrexial, the dyspnea, prostration and cyanosis are great, and the respiration rapid and more labored.

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[1] Pleuritis und Pneumonie im Kindersalter, Berlin, 1862, 339.

[2] Traité de mal. de l'enf., 1904, III, 397.

As pointed out elsewhere, the positive differentiation between a simple and a *tuberculous* bronchopneumonia is usually impossible except by the discovery of tubercle bacilli in the sputum procured by introducing a swab into the pharynx (see Vol. I, p. 560); and even this examination is far from conclusive if a negative result is obtained. Theoretically, the lesions are oftener in the upper lobe and in front in the tuberculous form, the course is slower, and the temperature less high.

In very young infants, and in older infants with narrow, deformed chests due to the presence of rickets, the diagnosis must sometimes be made between bronchopneumonia and *atelectasis*. The absence of a rapidly developing prostration and high fever indicates the latter condition; yet fever may be absent or slight in either disease. The deep and rapid respiration which may occur in *acidosis* is without the characteristics of actual dyspnea and is not attended by cyanosis.

The employment of the *x*-ray is of service in recognizing areas of consolidation in the lung. Its value must always be judged, however, in connection with the symptoms and physical signs of the case; and it does not differentiate the disease from tuberculosis or from croupous pneumonia.

Treatment. **Prophylaxis.**—Everything must be done to prevent the occurrence of the disease. Especially in infancy the danger of pneumonia developing subsequent to chilling must be guarded against. In general the prophylactic measures are to be followed recommended for preventing any inflammation of the respiratory tract. Association of infants with those suffering from acute catarrhal processes of the respiratory apparatus needs to be avoided, owing to the possible danger of infection and of its extension to the lungs. There seems no good evidence that primary bronchopneumonia is itself contagious, although that secondary to the infectious diseases, especially pertussis, diphtheria, and measles, would appear at times to be able to communicate itself readily to other children with these diseases. Consequently subjects in wards for infectious diseases, who develop bronchopneumonia, should preferably be isolated. Particularly is care to be taken to relieve as quickly as possible a bronchitis from which an infant chances to suffer, since this disease at this period of life is so frequently followed by pneumonia.

Treatment of the Attack.—Although various attempts have been made to establish a specific treatment for the disease by the use of sera, no certain remedy has as yet been obtained, and we are limited to treatment which is purely symptomatic. The researches of Cole and others (see p. 000) have succeeded in obtaining from inoculated horses a serum which is believed to possess a specific action in cases of pneumococcus infection of the Type I strain; but inasmuch as this variety is seldom the cause of pneumonia in infancy and childhood, it is of little aid in the therapeutics of the disease at this time of life.

Hygiene.—The patient will naturally be confined to bed. His position there should be changed frequently, laying him now upon the abdomen, now on one side or the other, in order to prevent the production of a hypostatic congestion. Infants may with advantage be taken up at times and held in the nurse's arms, and possibly dyspnea may be relieved if the infant's head is thus looking over her shoulder. In like manner older children may be relieved by extra pillows. All this is an individual matter and must be suited to the case; but frequent change of position of some kind is essential.

The open cold-air treatment, referred to also in considering Croupous

Pneumonia" (see p. 67), has come increasingly into vogue in recent years, and is undoubtedly of the greatest benefit in properly selected cases. There are, however, many exceptions to this, and, like methods of treatment of any sort, it cannot be used without discrimination. This is especially true of very young infants and of those in whom a bronchitis with abundant secretion constitutes the predominating element. Here the best results, in my experience, are obtained by placing the patient in a room with warm, moist air, and using the croup-tent when necessary. Just as the improvement and increased comfort is in many cases often very marked when the patient is placed in the open air, so in other cases it is surprising what relief to the cough, dyspnea, cyanosis and general distress may promptly follow treatment with a croup-tent. Only trial may serve in many instances to show which plan is more applicable. The cold-air treatment, although so often effective, would appear to have been based upon a physiological observation which has not been sustained by further studies, viz. that it increases blood-pressure and that this increase is a matter to be desired. The observations of Freeman[1] and of Morse and Hassman[2] and others, would indicate that the blood-pressure does not bear any relationship to the temperature of the surrounding air, or that it may even rise after the child has returned to the warm room; and it has been shown by others that it possesses little relationship to the prognosis. This does not, however, negative the undoubted improvement in the general condition which many children show through cold-air treatment. It is mentioned here for the purpose of warning against the acceptance of routine treatment of this or any sort. As the air under a croup-tent readily becomes overheated and lacking in freshness, the use of the tent should not be continuous. Always, too, in moist-room treatment or under any other conditions measures must be taken to insure that the air is pure. An open fire-place with fire burning offers one of the most satisfactory means for obtaining this, or two rooms may be used and the patient taken from one to the other after this has been well aired and warmed. In all cases which become protracted, and in those with comparatively little catarrhal secretion, the cold-air treatment should be tried.

Diet.—Milk is probably the best general food, but this need not be the only article of diet. The appetite is so diminished that there can be given any form of digestible liquid or semi-liquid nourishment suitable to the age, which the patient can be induced to take. This is of importance, as the disease is usually an exhausting one with a more or less prolonged course. The state of the digestion is sometimes such that undiluted cow's milk is not well tolerated. An abundant supply of water must be furnished, and if not enough liquid is ingested a daily enteroclysis is of value.

Counterirritation.—Treatment of this sort is of benefit especially in cases with a large bronchitic element. The frequently repeated application of mustard-plasters every 3 or 4 hours is one of the best remedies under these circumstances. This is useful, too, in instances of failing heart or respiration, but less so in urgent cases than is the mustard pack. (See Vol. I, p. 242.) The cotton jacket is in my opinion of little value. Poultices and similar applications are rarely useful, and often harmful through their weight and interference with the ease of respiration. Dry cups are often of service in severe cases.

Inhalations.—These are of great service in relieving cough. Benzoin and menthol may be used in the water employed in the moist-room treatment, or given from a croup kettle under a tent. Inhalations of oxygen, not too continuously employed, are sometimes of value in cardiac or respiratory failure. For children placed in the croup-tent the administration of oxygen may often be combined with that of the water-vapor, as tending to increase the vitality of the air in the tent. The results from oxygen are, however, on the whole disappointing, doubtless because in the cases in which the remedy seems indicated the condition is such that little benefit can be expected from any course of treatment.

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[1] Amer. Jour. Med. Sci., 1916, CLI, 1.

[2] Amer. Jour. Dis. Child., 1916, XII, 445.

Hydrotherapy.—This fulfills many purposes in the treatment of bronchopneumonia. In infants the warm tub bath (98 to 100°F.) (36.7° to 37.8°C.) given every 3 hours if the temperature of the patient reaches 103°F. (39.4°C.) or if there are decided nervous manifestations, often produces fall of temperature, stimulates the circulation, relieves restlessness and dyspnea and induces sleep. It is, I believe, one of the best of measures in the management of bronchopneumonia. In cases with very feeble circulation and in those suffering from bronchopneumonia with low temperature, brief plunging into the hot bath (105°F.) (40.6°C.) with or without mustard is sometimes helpful. The tepid pack (80° to 90°F.) (26.7° to 32.2°C.) followed by wrapping in blankets is often successful in the reduction of fever, and may be used in any case of hyperpyrexia in older children where the tub bath cannot be employed. Sponging is usually more tedious and trying to the child and less efficacious than the tub bath.

Many older children and most infants do not tolerate cold water well, and sponging or tubbing with water of 70°F. (21.1°C.) is seldom more efficacious and generally much more depressing than the use of warm water. Indeed, hydrotherapy in any form must be suited to the case, and abandoned if not well borne. All symptoms indicating cardiac debility are of especial warning against the use of cold water.

Internal Medication.—There should be no routine method in the use of drugs in this disease. In fact, they should be given only with care, when really needed, and with due consideration of the possible secondary evil effects particularly upon the digestive apparatus. At the same time when given at all the dosage should not be so small that it can serve no useful purpose. Moderate stimulation is advisable in many instances. It is easier to sustain the strength of the patient than it is to restore it after failure has taken place. Much has been written against the employment of alcohol, and there is no doubt that it has been used too frequently and when not indicated. My belief, from long-continued clinical observation, is that in some way it does good in many cases to an extent not attained by anything else. This by no means signifies that every case should receive alcohol. The initial dose should be small, 15 to 20 minims (0.92 to 1.23) of whiskey or its equivalent, every 3 hours for a child of 2 years, increasing the quantity if needed. The milder cases need no stimulation whatever. Tincture of digitalis is invaluable if cardiac failure threatens, as shown by the appearance of pallor or of slight cyanosis of the lips, cheeks and fingers. Its action is, however, slow. From 1 to 2 minims (0.062 to 0.123) every 4 to 6 hours for an infant of 1 year is a moderate dosage. For rapid stimulation in cases of sudden cardiac or respiratory failure reliance should be placed upon hypodermic injections of 2 to 4 minims (0.123 to 0.246) of camphor in olive oil (1 : 10), caffeine sodio-benzoate (⅛ to ¼ gr.) (0.008 to 0.016) or epinephrine (2 to 4 minims (0.123 to 0.246) of a 1 : 1000 solution) for a child of 1 year. Nitroglycerine hypodermically, 1⁄200 gr. (0.0003) for a child of 1 year, is often a valuable remedy in relieving an overburdened heart. Atropine given hypodermically is not only a serviceable respiratory stimulant, but, I think, very useful administered in cases where there is a large amount of secretion in the lungs. The dose may be 1/2000 to 1/1000 of a grain (0.00003 to 0.00006) every 3 to 4 hours for a child of 1 year, flushing of the face indicating that the remedy must be suspended or diminished in amount. Of strychnine I make but little use. It may be exhibited in doses of 1/300 of a grain (0.0002) every 3 or 4 hours at 1 year for cardiac debility, but offers no advantage over other drugs less likely to produce nervous excitability. What has been already said (p. 64), regarding the prognostic value of the blood-pressure, is to be borne in mind in the choice of the drugs mentioned; and none which have proven themselves clinically useful need as yet be discarded on account of their apparent physiological action as determined by animal experimentation.

The only sedative of value is opium, and this must be employed with great judgment, since it tends to interfere with respiration, to diminish too greatly the expulsive power of the cough, and to increase the tympanites. It is indicated in those cases where a racking, incessant cough is weakening the strength of the patient and preventing sleep, and is not relieved in other ways. Deodorized tincture of opium in doses of ½ to 1 minim (0.031 to 0.062) may be given to a child of 1 year, repeated as indicated; while for older children heroin is a useful sedative. An emetic is at times of value for patients in whom the lungs appear to be filled with secretion, interfering greatly with respiration. It is not often required. The coal-tar antipyretics in small doses (1 to 2 grains (0.065 to 0.13) of antipyrine or phenacetin for a child of 2 years) are often serviceable for relieving nervous symptoms, and may be employed, too, in those cases of hyperpyrexia in which hydrotherapy is not well tolerated. The dose may be repeated at frequent intervals until some effect is produced, but great care must be exercised not to give such an amount as will result in decided fall of temperature with sweating and depression. Their employment to reduce fever is seldom called for.

Special Symptoms and Complications.—The treatment of most of such symptoms has already been outlined in what has preceded. A few additional remarks may be in place, partly in the way of condensed review: Cyanosis and pallor demand stimulation. If sudden and severe, with collapse, the hot mustard pack or the mustard bath is indicated, oxygen should be given, and hypodermic stimulation must be vigorous. Fever requires no treatment if moderate (103 to 104°F.), (39.4° to 40°C.), except the employment of warm tub-baths. In fact, except in cases of hyperpyrexia, the fever does no harm, and its treatment is rather for the relief of the attending nervous symptoms. Excessive bronchial secretion is best treated by counterirritation and atropine. Expectorants of any sort are of doubtful value. Cough is often relieved by inhalations; if not, opiates in small dose and given carefully may be needed. Nervous symptoms are to be treated by hydrotherapy or by the administration of small repeated doses of antipyrine or phenacetin. Tympanites is to be relieved by the use of the rectal tube, douching the intestine with a saline solution, the employment of stimulants, the application of weak mustard-plasters to the abdomen and the administration, per rectum, of milk of asafetida. In severe cases, where respiration is greatly interfered with by the upward pressure of the accumulation of gas, a hypodermic injection of eserine, 1/500 of a grain (0.00013) to a child of 2 years, often gives great relief. It is, however, a depressing remedy. Diarrhea and vomiting may demand a change of the diet, especially a temporary withdrawal of milk. Douching of the bowel is also of benefit, as is frequently the administration of a cathartic. Convulsions occurring during the disease require bromides and possibly chloral.

Convalescence.—After the disease is over, patients often need tonic treatment, and especially change of climate to a region where the child can be nearly all day in the open air. This is especially true if decided debility remains or the physical signs are slow in disappearing.

CROUPOUS PNEUMONIA

(Lobar Pneumonia. Fibrinous Pneumonia)

For reasons already given "lobar" (see p. 48) is an unsuitable term, and of the others the word "croupous" has been by usage associated so long with the disease that it may well be retained. "Congestion of the lungs" is a title to be avoided since in its active form this condition is only the early stage of the pneumonic process.

Etiology.—The influence of age is an important predisposing cause; yet from the fact that children attacked recover in the large majority of cases, and for other reasons already presented (p. 47) statistics upon the matter are not reliable. The disease is in any event one of the most common affections in early life. Any age may suffer from it and even congenital cases with intra-uterine infection are reported by Netter,[1] Levy[2] and others. Yet it is much less often observed in the first 2 years of life, and especially in the 1st year, than is bronchopneumonia. It then increases in frequency, more cases occurring from 2 to 6 years of age than at any other period in early life. The statistics of Comby[3] in 356 cases in children give from 3 months to 2 years, 45 cases (13 per cent.); 2 to 5 years, 170 (48 per cent.); 5 to 10 years, 102 (29 per cent.); 10 to 15 years, 39 (11 per cent.). Holt and Howland[4] in 500 cases found in the 1st year 76 cases (15 per cent.); from 2 to 6 years, 309 (62 per cent.); from 7 to 11 years, 104 (21 per cent.); from 10 to 14 years, 11 (2 per cent.). Dunlop's[5] figures vary considerably from those given, principally with reference to the frequency in early childhood. Thus in 147 cases there were under 2 years, 45 (31 per cent.); 2 to 5 years, 42 (28 per cent.); 5 to 12 years, 60 (41 per cent.).

Sex is a much less important etiological factor in early life than it is in adults, although a somewhat larger number of males appear to be attacked. Season exerts a decided influence, the greatest number of cases developing in the colder months of the year and most of all in the spring. In Comby's series of 356 cases 138 (39 per cent.) occurred in the spring; 72 (20 per cent.) in summer; 57 (16 per cent.) in autumn and 89 (25 per cent.) in winter. The disease is widespread in the temperate zone but less common in the tropics.

Causes resulting in direct irritation of the mucous membrane appear to be important. Primarily here is exposure to chilling, the congestion of the mucous membrane which this produces allowing the germs of the disease to enter the lungs. In the same way trauma of the chest, as by a blow or by a fall from the bed or coach, is sometimes followed by the development of pneumonia. The previous health predisposes only to a limited extent. The great majority of cases are *primary* in origin and occur in previously healthy children. Although infants in a debilitated state of health may contract it, the disease is more liable in these subjects to be of the bronchopneumonic type. The only decided exception is tracheobronchitis, which may readily be followed by either bronchopneumonia or the croupous form. Even this condition, however, is oftener absent than present as a precursor of croupous pneumonia. The various infectious diseases, especially diphtheria, pertussis, grippe and measles, may not infrequently be accompanied by croupous pneumonia in older children, but bronchopneumonia is the variety oftenest seen. An earlier attack exercises a predisposing influence for a later recurrence of the disease, although this may be classified rather as an individual predisposition. Ziemssen[1] found 19 out of 201 cases of pneumonia in children in whom the disease had occurred previously, and in some of them more than once. So, too, a family predisposition exists and occasionally two children in a family will be attacked by pneumonia at the same time. Even the epidemic outbreak of croupous pneumonia has been reported by different observers but is unusual, and contagiousness exists to a very slight degree. This latter question has been studied by Netter.[2]

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[1] Soc. de biol., 1889, March 9, 187.

[2] Arch. exper. path., 1889, XXVI, 155.

[3] Traité des mal. de l'enf., 1904, III, 425.

[4] Dis. of Inf. and Child., 1916, 526.

[5] Brit. Med. Jour., 1908, II, 367.

Exciting Cause.—The disease is clearly an infectious one and is often with propriety classified by writers under infectious disorders. The first germ intimately associated with it in medical literature was the pneumobacillus of Friedländer,[3] but it has since been proven that the usual agent is that designated the diplococcus lanceolatus (diplococcus pneumoniæ) or pneumococcus, earlier described by others but claimed to be the cause of pneumonia by Fränkel[4] and by Weichselbaum.[5] With this may be associated the streptococcus, staphylococcus, influenza bacillus, typhoid bacillus, colon bacillus, pneumobacillus, or other germs; and exceptionally the Friedländer bacillus acting alone would appear to produce the disease. The researches of Cole, Dochez, Mitchell and others upon the different strains of pneumococcus, their frequency of occurrence and their prognostic influence have already been referred to in the section upon Bronchopneumonia (p. 50). In the 90 cases of pneumonia studied by Mitchell,[6] nearly all of them of the croupous form, approximately 75 per cent., showed the presence of Type IV of the pneumococcus. The pneumococcus is found in the affected lung, the sputum, and in complicating lesions such as pleurisy, pericarditis, meningitis, and peritonitis, and not uncommonly is discoverable in the blood. The germ probably makes its way directly from the nasopharynx to the lung, where it infects the mucous membrane which has lost its resisting power; or perhaps it enters the blood-vessels through the enfeebled mucous membrane of the nasopharynx.

Pathological Anatomy.—Opportunity to study the lesions in early life is not frequent owing to the low rate of mortality. The changes found are much the same as in adult life, with an increased tendency to bronchial catarrhal inflammation and to pleurisy. The basis of the process is a fibrinous inflammation occupying the alveoli and bronchioles. In the first stage, that of *congestion*, there is hyperemia of the affected area, rendering it edematous and dark-red in color. An exudation of serum and round cells into the alveoli begins and the lining epithelium is swollen. The stage of congestion lasts usually a few hours only, but may be prolonged several days. In the second stage, that of *red hepatization*, the affected pulmonary tissue is hard, swollen and on section cuts and looks like liver, but with a coarsely granular surface. The alveoli and bronchioles are filled with an exudation of fibrin, epithelial cells, pneumococci, and red and white blood-corpuscles, the fibrin predominating and being found in the lymph-spaces as well. There is usually a pneumococcic fibrinous exudate of varying thickness upon the pleura, perhaps only sufficient in amount to make the surface appear lustreless and rough. The duration of the second stage is variable, from several days up to 1 or 2 weeks. The third stage, that of *grey hepatization*, develops gradually from the second, the lung becoming more moist and paler from the presence of less congestion and through the increase of the exudation of white blood-cells which have undergone fatty degeneration. The pleural inflammation persists and sero-fibrinous or sero-purulent fluid may be found in the pleural cavity. The fourth stage, that of *resolution*, follows, the lung becoming softer and again containing air. The change is brought about by the liquefaction of the inflammatory products, which are for the most part absorbed by the lymphatic vessels and to a limited extent removed by way of the bronchi in the form of sputum. Resolution lasts but a few days as a rule; but sometimes is considerably delayed and may not be complete for weeks or months. (See Delayed Resolution, p. 84.) It generally begins with the fall of temperature to normal. In some cases the stage of grey hepatization is followed by the infiltration of the affected tissue with great numbers of pus cells, but this is uncommon.

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[1] Pleuritis und Pneumonie im Kindersalters, 1862, 153.
[2] Arch. gén de méd., 1888, CLXI, 530.
[3] Fortschr. d. Med., 1883, XV, 715.
[4] Verhandl. III, Kong. inn. Med., 1884, III, 17.
[5] Wiener med. Jahrbücher, 1886, 483.
[6] Journ. Penna. State Med. Assoc., 1917, XX, 343.

In cases where the consolidation has extended slowly not all portions of the lung show at autopsy the same stage in the pathological process; one region perhaps being merely congested, another about to undergo resolution, and a somewhat mottled condition results.

In addition to the pleura other organs than the lung often exhibit alterations. The bronchial lymph-nodes are swollen and the mucous membrane of the trachea and larger bronchi is generally reddened. Occasionally pericarditis, peritonitis or meningitis may occur. Rarely the lesions of arthritis, parotitis or general pneumococcic septicemia may be found.

Portions of the Lung Involved.—According to most investigators the disease appears to be localized oftenest either in the right upper lobe or the left lower lobe. As to the frequency in more detail statistics vary somewhat. Barthez and Sanné[1] in 408 cases found it in the right upper lobe 170 cases; left upper lobe 47; right lower lobe 41; left lower lobe 69. Comby's[2] figures based on 356 cases differ somewhat from these; viz. right apex 117 cases; right base 31; left apex 65; left base 75. In each series the remaining cases were distributed in various other parts of the lung. In Dunlop's[3] 147 cases the distribution was: left base 63 cases; right base 25; right apex 23; left apex 11; both lungs 8; whole of one lung 10. Koplik[4] in 217 cases found involvement of the right upper lobe in 74; right lower and middle lobes 49;

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[1] Traité des mal de l'enf., 1884, I, 712.

[2] *Loc. cit.*, 432.

[3] *Loc. cit.*

[4] Boston Med. and Surg. Jour., 1905, CLII, 741.

left upper lobe 35; left lower lobe 58. Generally, then, a considerable part of one lung is affected and the remaining pulmonary tissue is uninvolved: but in a varying percentage (Comby, 2 per cent.), (Jurgensen,[1] 9.76 per cent.). (Holt, 13 per cent.), (Dunlop, 5 per cent.) the pneumonia is double, portions of both lungs being diseased.

Symptoms. (1) TYPICAL COURSE.—In well-marked and what might be called typical cases the disease begins suddenly, usually with vomiting, accompanied by sudden rise in temperature and sometimes diarrhea. In infancy there is often an initial convulsion which replaces the chilliness of early childhood and the rigor occasionally seen in later childhood. The patient looks and feels ill. There is headache, prostration, flushed face, accelerated respiration and pulse, thirst, loss of appetite, and often pain referred to the chest or the abdomen. Cough may be frequent, dry, harassing and painful, or may be trivial. As the disease progresses the symptoms continue in force; the fever remains high; the tongue is coated; respiration may become more dyspneic, with moving of the alæ of the nose and a moaning expiration; the face is flushed; there is a slight cyanotic tint to the cheeks, lips and finger-tips: and later may follow more or less delirium, especially or solely at night, or stupor. No change takes place for the better, although often for the worse. The dyspnea perhaps grows greater, the pulse more rapid, and the strength less, with a decided degree of prostration and apathy; but by the end of from 5 to 7 days the temperature falls rapidly by crisis to normal, perspiration occurs, respiration becomes easy, all unfavorable symptoms promptly disappear, and convalescence is rapid. In some cases the fall of temperature is often accompanied by very profuse sweating, feeble pulse, coldness of the extremities, pallor, decided prostration, drowsiness, and sometimes signs of undoubted collapse. In cases which terminate fatally without crisis the respiration becomes more labored, and often finally shallow and very rapid; coldness of the extremities develops; there is cyanosis; rapid, feeble pulse, and increasing stupor and prostration.

A more detailed study of the characteristics usually met with is necessary:

Onset.—Prodromal symptoms lasting a few hours are sometimes observed; these consisting of malaise, headache, and chilliness. As a rule, however, such manifestations are absent or overlooked. An actual rigor is, indeed, the exception even in later childhood. Comby[2] observed it but 33 times in 356 cases. I have, however, seen a severe rigor in children under 6 years, and in infants very marked pallor and coldness may usher in the disease. The initial convulsion is not infrequent in infancy; less so in early childhood. Vomiting is one of the most common symptoms of the onset; but this usually ceases promptly.

Fever.—The rise of temperature is very rapid, reaching 103° to 105°F. (39.4° to 40.6°C.) within a few hours (Fig. 281). During the height of the disease there is little change in the elevation, the morning temperature being perhaps 1 or 2°F. (0.6 to 1.1°C.) less than that of the evening. The final fall is generally by a distinct crisis, a drop often of 6 or 8°F. (3.3° or 4.4°C.) to normal or below it occurring within the course of from 12 to 24 hours (Fig. 282). In exceptional instances the fall of temperature may be so great that symptoms of collapse may develop, and rarely even terminate fatally. The crisis takes place most frequently anywhere from the 5th to the 9th day, sometimes a day or two earlier or later. In the 147 cases in children studied by Dunlop the crisis occurred on the 2d day 2 cases; 3d day 3; 4th day 6; 5th day 16; 6th day 15; 7th day 16; 8th day 22; 9th day 14; 10th day 10; 11th day 7; 12th day 3; after the 12th day 6. In 132 cases with crisis reported by Schlesinger[1] the figures are very similar, viz. 2d day 1 case; 3d day 1; 4th day 13; 5th day 20; 6th day 30; 7th day 27; 8th day 15; 9th day 8; 10th day 10; 11th day 1; 12th day and later 3. Not infrequently shortly before the final disappearance of fever a *pseudo-crisis* occurs (Fig. 283); the temperature dropping to normal and the impression being given that the disease is over. A return to high fever on the next day does not necessarily mean an extension of the process in the lung. For some days after the crisis the temperature may remain subnormal. In infancy it not infrequently runs a more irregular course throughout the attack, with greater daily differences ex-

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[1] Ziemssen's Handbuch der spec. Path. u. Therap., 1874, V, 50.

[2] *Loc. cit.*, 433.

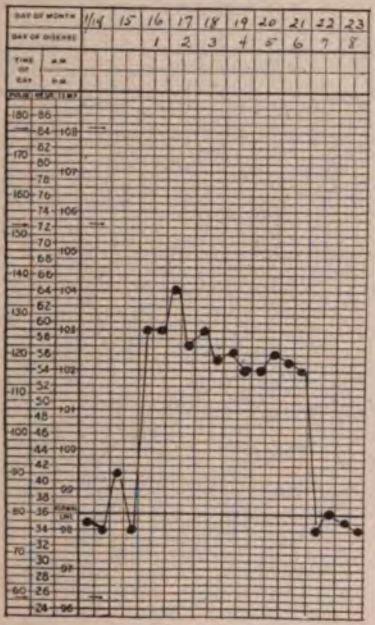

FIG. 281.

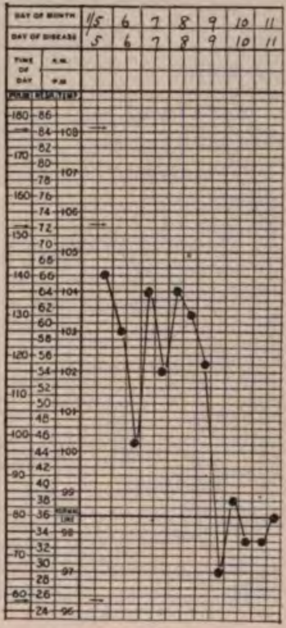

FIG. 282.

FIG. 281.—CROUPOUS PNEUMONIA, TYPICAL COURSE OBSERVED FROM ONSET.

Mary L., aged 1 year. Admitted to Children's Hospital of Philadelphia Jan. 11, for bronchitis. Coarse râles present throughout lungs; Jan. 14, lungs negative; Jan. 16, sudden rise of temperature, no physical signs; Jan. 19, impaired percussion and bronchial respiration at right apex behind; Jan. 21, distinct consolidation of right upper lobe, general condition good, pulse been 140 to 150 and respiration 50 to 65; Jan. 22, crisis.

FIG. 282.—CROUPOUS PNEUMONIA, SHOWING CRISIS.

Benjamin G., aged 5 years. Illness began 4 days previously, with cough, fever, loss of appetite; vomited on 2d day. Entered Children's Hospital Jan. 5. Examination showed dulness on percussion; and bronchial respiration at left base posteriorly and in axilla, a few râles; Jan. 10, signs clearing, bronchial respiration remains, general condition greatly improved.

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[1] Arch. f. Kinderh., 1897, XXII, 281.

hibiting almost a remittent type; and at this period of life a decline by lysis is frequently seen, the temperature descending gradually, requiring 3 or 4 days to reach normal (Figs. 284 and 290) and sometimes showing irregular elevations later. The remittent type of fever and the fall by lysis are not, however, uncommon in children of any age, and evening elevation for a day or two is not necessarily a matter of moment. Nevertheless a decided and more persistent return of fever after the decline renders one always suspicious of some complication, especially purulent pleurisy. (See p. 109, Fig. 301.) The relative frequency of fall by lysis is variously stated. Schlesinger[1] found it in 12 per cent. of 151 cases of pneumonia and Henoch[2] in 10.6 per cent. In fatal cases elevation of temperature sometimes increases toward the end of the attack and the patient dies with hyperpyrexia. However, the temperature in children is usually

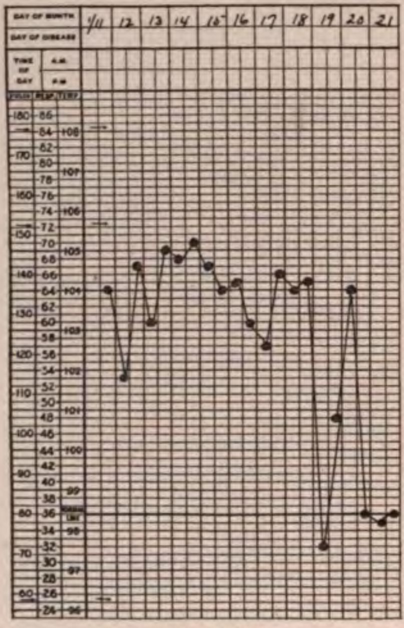

FIG. 283.

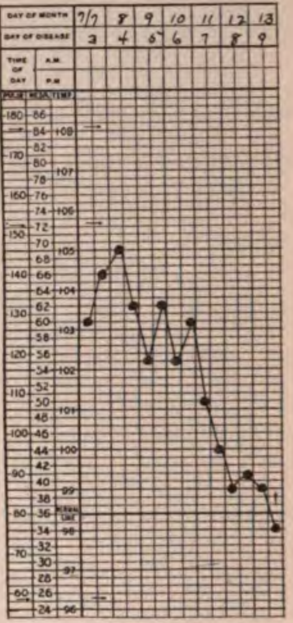

FIG. 284.

FIG. 283.—CROUPOUS PNEUMONIA, PSEUDO-CRISIS AND CRISIS.

Rosie L., aged 3 years. Previous duration of the disease uncertain. Been suffering from cough, fever, loss of appetite and occasional vomiting. Entered Children's Hospital Jan. 11. Examination showed bronchial respiration and dullness on percussion over the upper part of the right lung posteriorly; Jan. 16, some extension of the process in the right lung, respiration 40–60; Jan. 19. pseudo-crisis; Jan. 20, no change in general condition, many râles at right base; Jan. 21, crisis.

FIG. 284.—CROUPOUS PNEUMONIA, FALL BY LYSIS.

Walter E., 3 years old. Admitted to the Children's Hospital of Philadelphia, July 7, 1917. Illness began 3 days ago with vomiting. Dry cough on 2d day of disease. On admission delirious, toxic, apparently very ill, dulness, bronchial breathing and bronchophony right upper lobe. Severe symptoms began to ameliorate when the fall of temperature commenced.

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[1] *Loc. cit.*, 280.

[2] Vorlesung. ü. Kinderkr., 1895, 370.

greater than in adults, and a continued fever of 105° or even 106°F. (40.6° or 41.1°C.) is not in itself an unfavorable symptom. Recovery may follow even after a temperature reaches as high as 109°F. (42.8°C.) as in a case reported by Couch.[1] A similar case was observed by myself, the temperature dropping in 12 hours from 109°F. (42.8°C.) to nearly 97°F. (36.1°C.) (Fig. 285). It is to be noted in this connection that pneumonia, typical in other respects, may occasionally run an entirely afebrile course. Such cases have been reported but must be very uncommon. In other instances the temperature curve may be of an intermittent type.

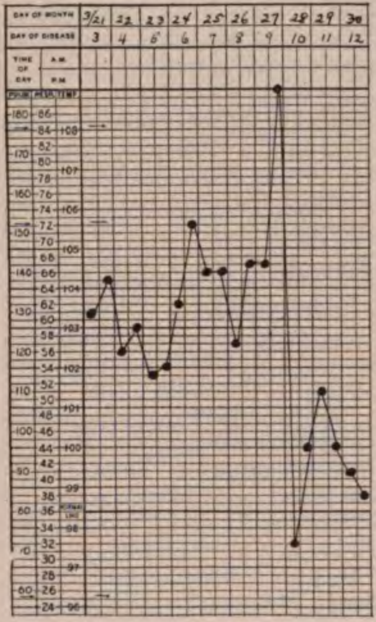

FIG. 285.—CROUPOUS PNEUMONIA WITH GREAT HYPERPYREXIA. CRITICAL FALL OF TEMPERATURE OF NEARLY 12° F. IN 12 HOURS.

Nathan M., aged 5¼ years. Typical croupous pneumonia upper portion of right lung. What appeared to be crisis occurred on the 10th day, with a drop of nearly 12°F. (6.7°C.) in 12 hours, with profound prostration, delirium and sleeplessness. This was followed by a slight extension of the lesion to the lower part of the right lung and moderate brief return of fever, the upper part of the lung having meanwhile cleared. Recovery.

Respiration.—The respiration is always increased in frequency, reaching from 40 up to 80 per minute. A certain degree of dyspnea is nearly always present, shown perhaps as mere irregularity and jerkiness in breathing. In the mildest cases it may be discoverable by placing the hand upon the abdomen, or by noticing the moving of the alæ of the nose, seen only when the patient is disturbed. In the majority of cases, however, this symptom is constantly present, although less intense than in bronchopneumonia, and is accompanied by inspiratory retraction of the costal interspaces, more or less tugging of the sternocleidomastoid muscles, and sinking in of the epigastrium and the episternal notch. In severe cases this respiratory involvement is excessive and every breath seems drawn with great voluntary effort. The expiratory moan accompanying respiration is a very characteristic symptom observed in many cases of average severity. The ordinary rhythm of respiration may be altered, the pause which normally follows expiration being transferred to the moment after inspiration.

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[1] Brit. Med. Jour., 1896, II, 1212.

Pulse.—This is always accelerated but of good quality and normal tension in favorable cases. In those of a more serious nature the tension is lowered and the pulse becomes more rapid, weak and running. There is cyanosis and distention of the veins of the neck. A pulse-rate of 140 to 180 is not infrequent in infants and young children, and is in itself not unfavorable if the character remains good.

Pulse-temperature-respiration Ratio.—Reference has already been made to the conditions obtaining in children in health (Vol. I, pp. 54, 56, 63). In pneumonia the pulse reaches a greater rapidity than in adult life, but allowing for the normally higher rate in a state of health at this time of life, from which the increase in the number of pulse-beats must, of course, be counted, it cannot be said to increase with any greater ratio to the increase of temperature (10:1) than occurs in adults. In fact in many cases the rate of increase is less than the adult ratio. The respiration on the other hand departs from the normal ratio to the pulse, and frequently equals that of 1:2½ or even 1:2. During convalescence the pulse regains its normal rate or is even for a time slower and irregular. This irregularity is not an unfavorable symptom.

Cough.—This is a most uncertain symptom in pneumonia in early life. Repeatedly cases occur in which there is no cough, or so little that it is overlooked. Generally, however, it is present and occasionally is distressing, and may be debilitating through the resulting lack of rest. This is far less frequent than in adult life. The cough is dry, short and often painful; consequently the patient makes an effort to suppress it, and a facial expression of pain, or a short cry, follows an attack. After convalescence begins the cough becomes looser through the establishing of secretion.

Expectoration.—In children beyond the age of 7 or 8 years, although not always then, sputum is expectorated having the usual rusty or blood-stained character and containing pneumococci. In infancy and early childhood there is rarely any expectoration, unless perhaps expelled as a result of the straining efforts attending vomiting.

Pain.—Thoracic pain is often present on coughing and often, too, independently of this. It is seldom as severe as in adults. Not infrequently the pain is referred to the abdomen, and when in the region of the appendix may cause great diagnostic difficulty. (See Vol. I, p. 808.)

Cry.—It has been claimed that children with pneumonia cry but little owing to the dyspnea present, with inability to sustain a prolonged sound. This is undoubtedly true in well-marked cases; but in milder attacks in infancy the cry is often loud and continued. The expiratory moan and the short cry after coughing have already been referred to.

Blood.—A prominent characteristic of pneumonia is the marked increase in the number of leucocytes which reaches 15,000 to 25,000, or more to the cubic millimetre, 50,000 or 60,000 being sometimes observed. (Average of 24 cases in infancy, 31,700; Heiman.)[1] The polymorphonuclear cells are those especially affected. Only in very mild cases, or in those unfavorable in which the organism shows no resisting power, is there little or no increase seen. The leucocytosis appears early and grows steadily greater until a day or two before the temperature falls. With the decline the number of cells decreases rapidly or slowly in proportion to the rapidity of the fall of temperature. If the temperature descends quickly and the leucocytosis persists it is probable that a pseudo-crisis has occurred. A slow decline in the number of leucocytes with a persistence of high fever indicates delayed resolution or the development of complications (J. Hess).[1]

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[1] Arch. of Ped., 1905, Oct.

Digestive Symptoms.—The initial vomiting seldom continues after the first day or two, but occasionally becomes a serious symptom. The appetite is greatly diminished or lost, although the active thirst may sometimes obscure this fact in the case of infants; milk being taken in place of water in order to satisfy the thirst. The tongue is coated but seldom dry except in severe cases; the throat is not often involved; constipation is frequent; diarrhea uncommon. Unusual tympanitic distention is a symptom of very unfavorable prognostic import, indicating the lack of resisting power of the muscles and adding greatly to the dyspnea.

Urine.—This is high-colored, of high specific gravity, scanty, and loaded with urates, as is febrile urine from other causes. The sodium chloride is markedly diminished, but becomes of normal amount at the time defervescence occurs. A febrile albuminuria with a few hyaline casts is not uncommon, but nephritis is unusual. The presence of acetone in small amounts is a matter of no moment.

Nervous Symptoms.—The pain in the chest and abdomen and the occurrence of initial convulsions have already been referred to. Headache and pain in the limbs may also occur as early symptoms. There may be delirium, perhaps only at night or while the temperature is increasing; but infants and young children do not often in average cases exhibit more disturbance than a degree of apathy and somnolence, or perhaps sleeplessness or great restlessness. The latter may reach active jactitation, especially if the dyspnea and consequent air-hunger are excessive. An unusual development of nervous symptoms is seen in the cerebral pneumonia to be described (p. 80). My experience agrees with that of others who have found no special relationship between the degree of nervous involvement and the portion of the lung affected. The severity of these nervous symptoms depends more upon the height of the temperature, the severity of the infection, and the general constitutional disturbance than upon the part or amount of pulmonary tissue affected. The patellar reflex is frequently absent (Pfaundler).[2]

General Condition.—There is little loss of weight in cases of average duration, despite the diminished amount of nourishment taken. There is, however, a very characteristic prostration. In many instances the decision against the presence of pneumonia can almost be made by merely observing the patient, who does not look sufficiently ill. A child with pneumonia has no desire to be out of bed and takes little, if any, interest in toys. Placing in the sitting position for examination causes evident fatigue if at all prolonged, and in many cases must be avoided entirely. The prostration is, however, only temporary, for as soon as defervescence occurs strength is regained with great rapidity and long confinement to bed during convalescence is not necessary in early life.

Skin.—Flushing of the cheeks is generally present, sometimes one more than the other; but this difference is without significance as indicating the portion of lung involved. Cyanosis may develop but in cases of moderate severity is slight and seen only as a faint purplish tint to the flushed cheeks or a slight blueness of the hands or feet. This degree of cyanosis is not a sign of immediate danger, but a warning that the respiratory or circulatory power is not entirely sufficient for the work demanded. Increasing cyanosis is a distinctly threatening condition, especially if combined with pallor. Herpes of the lips, face, or other region of the body is of frequent occurrence but probably not more so than in adults. It appears about the 3d or 4th day of the disease.

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[1] Amer. Jour. Dis. Child., 1914, VII, 1.

[2] Münch. med. Woch., 1912, XLIX, 1211.

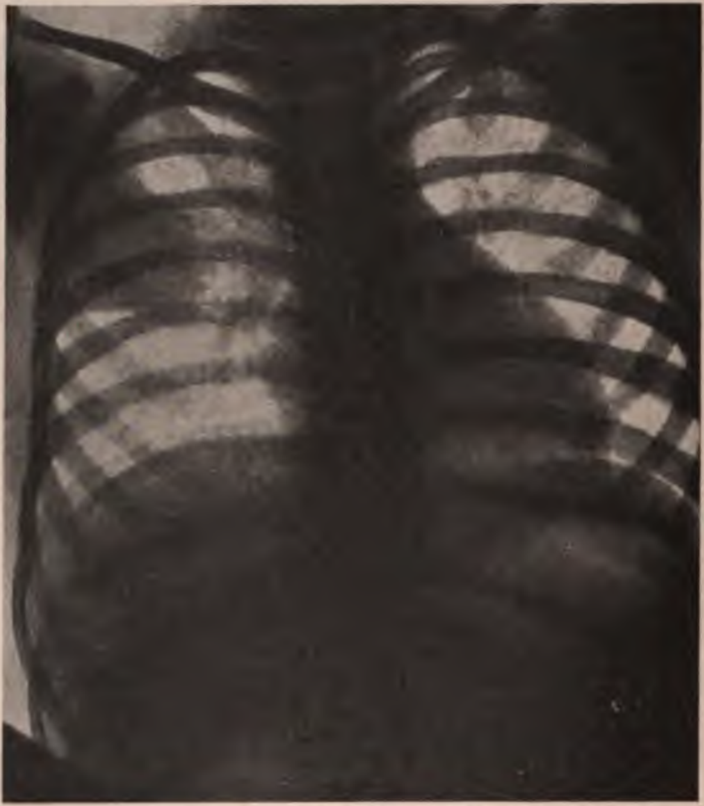

FIG. 286.—LOBAR PNEUMONIA.

5th day of the disease. No dulness and no bronchial voice or breathing. Radiograph shows cone-shaped consolidation beginning at the periphery and not reaching the root of the lung. (*Mason, Amer. Jour. Dis. Child.*, 1916, *XI*, 189.)

Physical Signs.—In cases exhibiting these in what may be called a typical manner the signs vary with the stage of development of the lesions. Early in the attack the most characteristic physical signs are crepitant râles at the end of inspiration, and slight localized impairment of the percussion note; or in some cases a tympanitic note in the region which will later reveal dullness. It must be stated, however, that truly crepitant râles are seldom discovered, the sounds being rather of a subcrepitant ure. Auscultation may show only slight prolongation of expiration a suspicion of a bronchial character, and slight increase of vocal resonance; or, on the other hand, feebleness of respiration over the affected area, possibly due to choking of the bronchus with secretion, with exaggerated breath-sounds over the healthy lung which is doing compensatory duty. As the consolidation increases and the affected portion becomes airless, perhaps by the 2d or 3d day, the respiration is distinctly bronchial; there is bronchophony; any mucous râles present are of coarse quality and appear to be very close to the ear; friction sounds may be heard; and the dullness on percussion is decided, although not often as great as in pleural effusion. Not infrequently the surrounding healthy region exhibits a hyperresonant note on percussion. If much of the lower part of the lung is involved the affected side of the chest may exhibit diminished expansion and less depression of the interspaces with inspiration. Palpation may reveal increased vocal fremitus, but the feebler, higher-pitched voice of children usually renders this sign of little value.

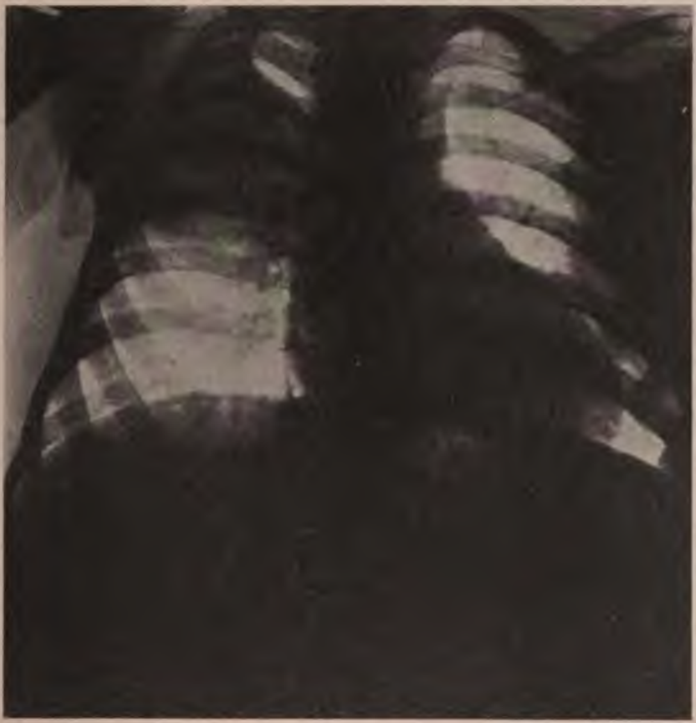

Fig. 287.—Lobar Pneumonia.

8th day of the disease. Dulness and bronchial breathing and voice. Radiograph shows consolidation extending from periphery to the root of the lung. A different patient from Fig. 286. (*Mason, Amer. Jour. Dis. Child.*, 1916, *XI*, 194.)

As resolution begins râles become more numerous and there is a disappearance of the evidences of consolidation. This disappearance is sometimes remarkably rapid, and even in average cases all or nearly all evidences have vanished in 2 or 3 days. Sometimes, however, a much longer time is required. (See Delayed Resolution, p. 84.)

The employment of the x-ray for diagnostic purposes not only confirms the presence of consolidation as determined by auscultation and percussion, but will not infrequently reveal it before it is discoverable by these means. This method of examination has apparently disproved the existence of central pneumonia, in which no physical signs are discoverable. Weill and Mouriquard[1] and Mason[2] have shown with the x-ray that pneumonia never begins in the centre of the lung, but always at the periphery, the early shadow being triangular in shape with the basis on the pleura. It is only when the apex of the consolidated portion has extended to the hilus of the lung that bronchial breathing and bronchophony develop (Figs. 286 and 287). Earlier than this only a slight impairment of percussion resonance and feebleness of respiratory murmur may be discovered, or often nothing whatever abnormal.

Significance of and Variation in Physical Signs.—The description just given is what may be considered the type; but owing to the great number and variety of the alterations observed, it cannot apply to the signs as usually encountered; nor are the indications of these always clear. One of the great characteristics of pneumonia in children is the frequent lack of correspondence between the severity of the symptoms and the degree of development of the physical signs. This is particularly true of infancy and early childhood, at which period the results of physical examination are especially uncertain. In fact, at least early in the disease, the symptoms may clearly indicate the existence of a pneumonia while the signs are inconclusive. The dullness on percussion is often absent during the whole of the attack, and signs of any nature may be discovered only just before or at the time of defervescence. The hyperresonant or tympanitic note frequently heard may denote the existence of healthy lung doing compensatory work; but not uncommonly may indicate that consolidation is about to develop in the region where the note is audible. In the latter event the percussion is often deep and full—not the high-pitched Skodaic tympany—and may sometimes be obtained even in regions where auscultation shows distinct evidences of consolidation. Later the tympany is commonly replaced by dullness. When a tympanitic note is heard under the clavicle it often signifies that consolidation is to be looked for posteriorly at the base or elsewhere on the same side. The superficial breathing of children with pneumonia frequently renders auscultation valueless and deceptive. A lung may appear normal until the child, as a result of crying, coughing, or other cause, draws a deep breath, when the bronchial character of the respiration may become evident. In other cases possibly a slight roughening of the breath-sounds or a little prolongation of expiration may be all that can be detected. In many instances the auscultation of the cry is of far more value than that of respiration; bronchophony being perhaps revealed by it in a small area otherwise without physical manifestations. In fact the crying of the patient during the examination is sometimes rather to be desired than considered disturbing; for after the cry deep inspiration follows, and in place of expiration vocal resonance may be studied to advantage. In other instances the persistence of localized râles is sufficient, with the general symptoms of the disease, to make the diagnosis of consolidation of this region certain; and this is especially true if the râles appear to be very near the ear; *i.e.* there is *increased vocal resonance of the râles*. Crepitant râles are much less common in early life than in adults, being replaced by those of a more moist character.

One of the most frequent mistakes is the incorrect diagnosis of pneu-

[1] Ann. de méd. et de chir. inf., 1913, XVII, 275.

[2] Amer. Jour. Dis. Child., 1916, XI, 188.

monia upon the wrong side of the chest. This occurs early in the attack and is the result of comparing feeble respiration of the affected side with the exaggerated breathing of the other side, the incorrect conclusion being drawn that the latter is bronchial in character. Another mistake easily made is that of diagnosing the presence of a double pneumonia when the sounds heard upon one side of the spinal column are merely those transmitted from the thoroughly consolidated lung upon the other side; the small size of the chest in early life readily lending its aid to this error.

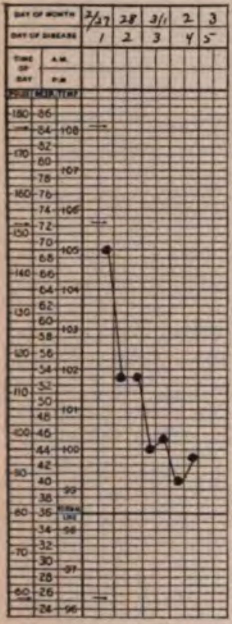
FIG. 288.

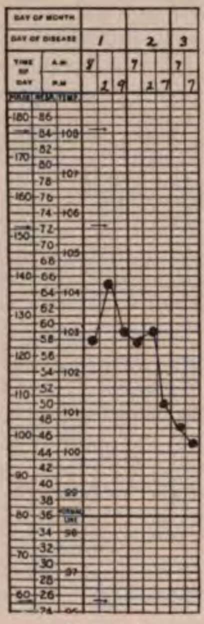
FIG. 289.

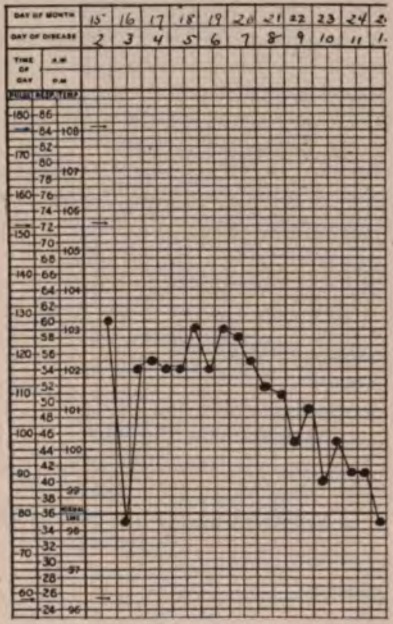
FIG. 290.

FIG. 288.—CROUPOUS PNEUMONIA, ABORTIVE TYPE.

Marina W., aged 10 years. Onset with chill, rapid respiration. Consolidation of left base on 2d day. Rapid recovery.

FIG. 289.—CROUPOUS PNEUMONIA, ABORTIVE TYPE.

A. A., male, 1 year old. Sudden onset, with cough, evidently severe illness, respiration dyspneic, from 60 to 70. Rapid, short course, child convalescing on 2d day. No certain physical signs found until several days after attack was over.

FIG. 290.—CROUPOUS PNEUMONIA, CEREBRAL TYPE.

Ambrose G., aged 5½ years. Onset with vomiting and headache. Delirium most of time, alternating with dulness. Supposed to be typhoid fever or tuberculosis. Very little cough. Physical signs first found on 8th day at right apex, fall by lysis.

It is often necessary to examine the lungs carefully day after day before a diagnosis based upon physical signs can be made; not forgetting in the search careful examination of the apices in front and of the axillary regions. In these cases the lesion must be either very small or so situated that it cannot be recognized by the ordinary methods. The importance of light percussion in pneumonia, using a single finger, cannot be too strongly insisted upon. By using a forcible tapping all abnormal percussion-signs are concealed by the general vibration of the whole resilient chest characteristic of the child.

Variations from the Type.—Certain of the variations from the normal course are so characteristic that they constitute distinct varieties of the disease. Among these the following are to be considered in addition to the typical course described.

2. Abortive Pneumonia.—This not infrequent form begins with the same suddenness and intensity as characterizes the more ordinary cases. The symptoms are well developed, but the process terminates abruptly after only 3 or 4 days illness, or even less than this, and in cases of very short duration the physical signs may not become evident. Among the abortive cases are to be included many of those called "congestion of the lungs," the pathological process reaching only this stage; and very probably many other instances of high fever of short duration in childhood, in which the diagnosis was entirely unsuspected. In other cases the physical signs of consolidation become more or less evident, but perhaps only after fall of temperature has occurred. Usually there is then a rapid disappearance of the physical signs of consolidation, although sometimes they are more persistent. A number of interesting instances of this type have been detailed by Kerr,[1] and I have witnessed the condition repeatedly (Figs. 288 and 289).

3. Cerebral Pneumonia.—The well-recognized variety with this title is characterized by an unusual prominence of cerebral symptoms (Fig. 290). These may be present from the onset or develop during the course of the disease. Not only do convulsions occur as an initial manifestation, but these may perhaps be repeated during the course of the attack. In other cases there may be unusually severe headache, squinting, rigidity, opisthotonos, persistent active delirium, coma, repeated vomiting, or other symptoms suggesting meningitis. When the physical signs are late in appearing diagnosis may be very difficult. In fact, a serous meningitis actually is present in many cases, the cerebrospinal fluid showing an increase of pressure, and the number of cells being above normal. In many others, however, the condition is purely a toxemic one. (See Meningismus, p. 316.) There seems to be no good ground for the formerly prevalent belief that pneumonia of the apex was particularly prone to exhibit cerebral symptoms.

4. Gastric Pneumonia.—In this variety the digestive apparatus appears especially involved. The tongue is coated, the appetite lost, diarrhea is common, and vomiting occurs early and may be persistent without the presence of cerebral symptoms to suggest meningitis as the cause. Many such cases continue for days without physical signs, and give many reasons to believe that the attack is due to a digestive disturbance.

5. Appendicular Pneumonia (Figs. 291 and 292).—The characteristic of this variety is the unusual degree of pain and tenderness in the appendicular region, while symptoms pointing to respiratory disorder are but little marked. Many such cases have been wrongly diagnosed as appendicitis, and operations have repeatedly been performed, and the discovery of a normal appendix made. I have previously[2] reported a number of instances and reviewed the published cases. Since then an extensive literature has accumulated, and I have seen the condition many times. (See Vol. I, p. 808.)

6. Wandering Pneumonia. (*Prolonged Pneumonia*).—Instead of terminating at the usual time such cases are characterized by an exten-

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[1] New York Med. Record, 1910, LXXVII, 701.

[2] Jour. Amer. Med. Assoc., 1903, Aug. 29, 531.

sion of the pneumonic process from one portion of the lung to another. The temperature may be persistently elevated or there may be repeated remissions, or even a crisis, to be followed immediately by a renewed elevation marking the development of the disease in a fresh location. In the meanwhile resolution may take place in the part first affected and the physical signs disappear there. (See Fig. 285, p. 73.)

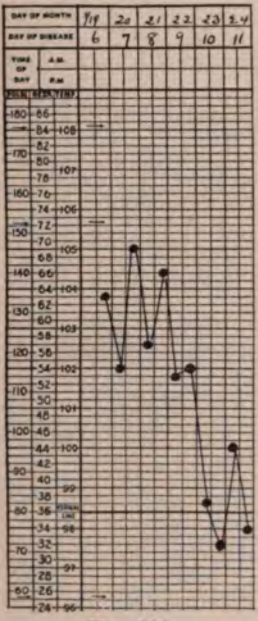

FIG. 291.

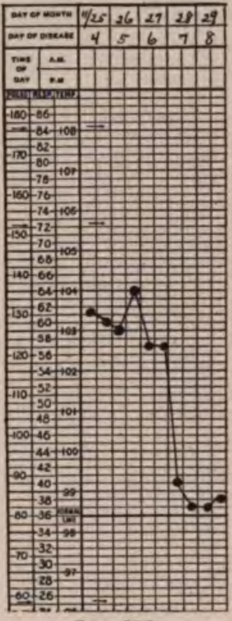

FIG. 292.

FIG. 291.—APPENDICULAR PNEUMONIA.

Simon L., aged 4 years. Taken ill acute Jan. 14, with obstinate constipation, high fever, and abdominal pain and distention in right iliac fossa. No cough noted. Diagnosis of appendicitis made by attending physician. Operation refused by parents. Admitted to Children's Hospital of Philadelphia, evening of Jan. 19. *Examination Jan. 20.*—Child in severe pain, anxious expression of face, thighs flexed on abdomen, respiration shallow, 30 to 40, high fever, abdomen rigid everywhere and tender in region of appendix, lungs in front negative. On account of distress of child and apparent certain diagnosis, no examination made of back on this date; Jan. 21, pneumonic consolidation left lung undoubted, abdominal symptoms unchanged; Jan. 22, abdominal symptoms gone; Jan. 23, crisis.

FIG. 292.—APPENDICULAR PNEUMONIA.

Walter E. C., aged 11 years. Admitted to Children's Hospital of Philadelphia, Nov. 25. Taken ill acutely Nov. 22, with vomiting, headache, and pain at McBurney's point. This very severe and continuous until admission. Case pronounced appendicitis by two prominent physicians, one a surgeon. *Examination in Hospital.*—Respiration 40 to 45, fever, pulse 95 to 130, well-developed croupous pneumonia at base of right lung, pain had shifted to right hepatic region, epigastrium and right shoulder.

7. PLEUROPNEUMONIA.—A certain amount of pleural involvement is present in a large proportion of all cases of croupous pneumonia. The title pleuropneumonia is applied to those in which a very decided degree of a complicating pleurisy modifies the complex of symptoms. The constitutional manifestations are generally more severe and the pain in the thorax often more marked than in ordinary pneumonia. The physical

signs are a combination of those of pneumonia and of pleurisy. The presence of a plastic pleuritic exudate frequently produces numerous friction sounds early in the case, and this, or the development of effusion, later diminishes the intensity of bronchial respiration and bronchophony, and increases that of the percussion dullness, which persists for some time after the sypmtoms of pneumonia are over. Ordinarily the pleuritic condition does not go beyond the deposit of a large, thick

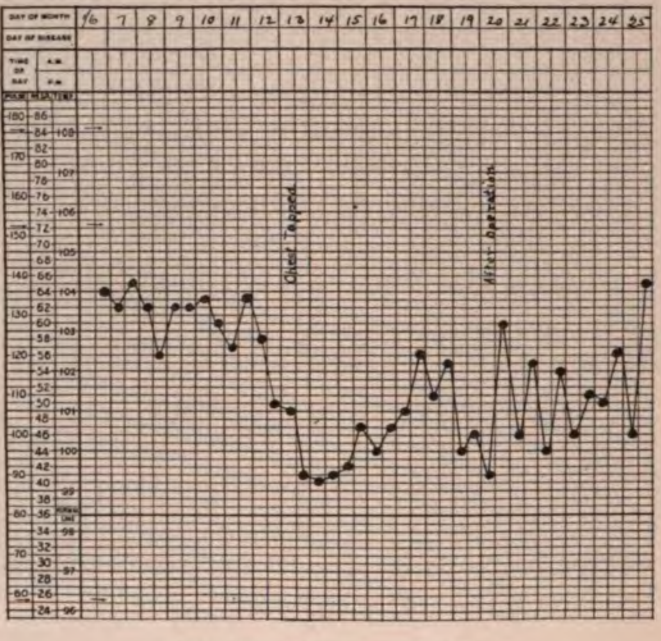

FIG. 293.—PLEUROPNEUMONIA.

Chas. E., aged 3 years. Admitted to Children's Hospital of Philadelphia, Jan. 6, on the 6th day of the attack, with well-marked physical signs of pneumonia in the upper part of the left lung in front and over the whole left side posteriorly; Jan. 10, whole left side very dull, expansion, fremitus and breath-sounds diminished, heart displaced; Jan. 13, aspiration gave 285 c.c. (9.64 fl.oz.) of straw-colored fluid containing staphylococci, temporary relief of symptoms with fall of temperature followed; Jan. 20, temperature soon rose again, general condition grew worse, very toxic, evident pressure-symptoms, exploratory puncture on 19th gave somewhat thicker and whiter fluid which deposited some sediment of pus-cells, transferred to surgical ward, and resection with drainage done; Jan. 25, been a profuse discharge of fluid, never of the characteristic appearance of empyemic pus as usually seen. Condition grew worse and death on this date.

plastic exudate with small localized foci of pus (Fig. 297, p. 101); but the development of a large empyema is very liable to take place in cases at all prolonged. Pneumococcic meningitis or peritonitis may be present also. (See also Pleurisy, p. 99.)

Complications and Sequels.—By all odds the most frequent complication of pneumonia is *pleuritis*. A dry pleurisy is in fact a nearly constant attendant upon all cases of pneumonia; or there may be a small amount of serous effusion. Large serous effusions are uncommon complications in early life, and particularly until later childhood is well ad- d, but the development of an *empyema* is a sequel especially to be before this age.

This occurrence is of much greater frequency in children than in adult life, and Gossage[1] observed it in 12 per cent. of 759 cases of croupous pneumonia in children. *Bronchitis* is frequent as a precursor of pneumonia, or as a complication in the part of the lung involved in the pneumonic process, and may seriously interfere with recovery in the case of infants. *Otitis* is also common, seen oftenest in infancy and early childhood. Schlesinger[2] observed it in 10.4 per cent. of his 173 cases, and Morse[3] in 17.8 per cent. of 100 cases of croupous pneumonia in infancy. *Endocarditis* and *pericarditis* are unusual. The latter is either fibrinous or purulent, and occurs oftenest in infants. *Peritonitis* likewise is not often seen. A *pneumococcic meningitis* is sometimes encountered and ends fatally. It develops usually late in the course of the pneumonia and is ushered in by convulsions, repeated vomiting and high fever, and the discovery of the pneumococcus in the spinal fluid obtained by puncture. It is distinctly uncommon. A *serous meningitis* is oftener seen, and systematic lumbar puncture in cases with meningitic symptoms might show it to be more frequent than ordinarily supposed. *Nephritis* occurs in occasional cases and perhaps more frequently than ordinarily believed; and *pharyngitis* is also encountered and may be pseudomembranous. Among rare complications reported may be mentioned arthritis,

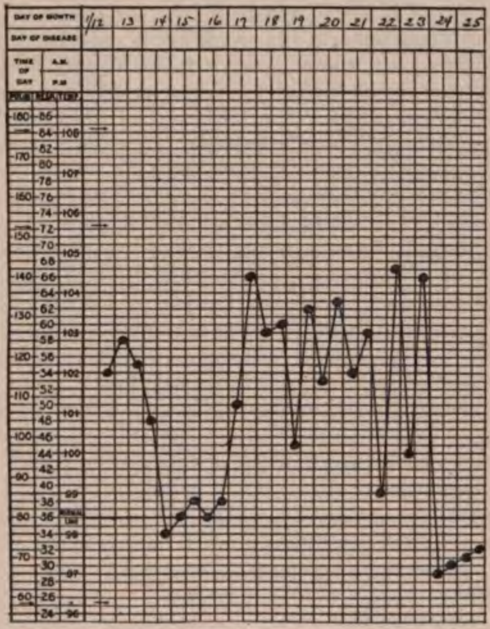

FIG. 294.—CROUPOUS PNEUMONIA WITH RELAPSE.

Jacob K., aged 3 years. Entered Children's Hospital of Philadelphia, Jan. 12, on the 5th day of the disease. *Examination.*—Lower part of lung on right side entirely consolidated; Jan. 13, crisis occurred with relief of symptoms; Jan. 17, after an interval of 3 days the disease recurred at the right apex and upper axilla; Jan. 24, second crisis on this date. Recovery.

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[1] Proc. Royal Soc. Med., 1907–8, I, Med. Sect., 63.

[2] *Loc. cit.*

[3] Arch. of Ped., 1904, Sept.

parotitis, multiple neuritis, osteomyelitis, subcutaneous emphysema, gangrene of the lung, pulmonary abscess, and general pneumococcic infection.

Relapse and Recurrence.—Chiefly to be distinguished from the extension of the process as observed in the cases of wandering pneumonia described, is the true *relapse* of the disease not infrequently seen, occurring after the attack appears to be over (Fig. 294). This may take place in another part of the lung, or there may be a re-development of the process in the portion which has just completely recovered. As to *recurrence*, one attack of pneumonia offers no protection against following ones; in fact appears rather to predispose to the development of the disease again at a later period. (See p. 68.)

Course and Prognosis.—The course of uncomplicated fibrinous pneumonia in children is generally severe, but the termination in those past infancy is, as a rule, favorable. After the fall of temperature, resolution of the lung takes place quickly and in a very few days all physical signs have disappeared. Sometimes the change from day to day is surprisingly rapid, but where any decided degree of pleurisy has been present the impairment of percussion resonance may be long in disappearing. Not infrequently, too, *delayed resolution* is observed. In these cases the patient, usually past the age of infancy, may continue to show both symptoms and physical signs of consolidation much longer than the usual time, or oftener may be entirely free from the former, but still exhibit the latter which finally disappear with remarkable slowness, sometimes continuing with little change for weeks or even for months.

The recovery of the general health and strength in most cases of pneumonia is very rapid. Appetite returns in full and the child is able and anxious to be out of bed. In fatal cases death takes place often from cardiac weakness with secondary failure of respiration, but not often from interference with respiration by the extent of the pneumonic infiltration. The presence of any degree of cyanosis is a distinct warning; and should this be marked and combined with pallor and rapid, weak pulse the outlook is unpromising. In cases nearing a fatal termination there are in addition to these signs coldness of the extremities; rapid, shallow, labored respiration; numerous coarse râles in the chest, and great prostration. Mere rapidity of the pulse in pneumonia is not of moment, unless there are other threatening symptoms present. Great abdominal distention by gas is also a distinctly unfavorable sign, as is a low leucocyte count, or unusually low blood-pressure. The significance of the blood-pressure has received considerable attention in recent years. The claim has been made that a low blood-pressure is an unfavorable symptom, and a high one favorable. While this is probably true within certain limits, it is by no means always so, as has been shown by the investigations of Porter and Newburgh;[1] Lambert[2] and others. (See also p. 64.) Early vomiting is a matter of no consequence, but this or diarrhea occurring later in the attack diminishes the chance of recovery. The mere existence of high temperature of 104° to 105°F. (40° to 40.6°C.) or over is not in itself an unfavorable prognostic indication in uncomplicated cases. Its persistence beyond the time when resolution should take place points to the possibility of some complication existing. Severe nervous symptoms early in the disease will probably be of temporary duration; occurring later they are of more serious prognostic import as they may depend

[1] Boston Med. and Surg. Jour., 1914, CLXX, 125.
[2] Jour. Amer. Med. Assoc., 1911, LVII, 1827.

upon a meningitis. The existence of any complication adds more or less to the gravity of the prognosis. Pneumococcic meningitis is usually fatal; peritonitis is very serious; bronchitis in rachitic infants impairs greatly the respiratory functions. An irregular continued fever beginning a day or so after the crisis renders the presence of an empyema very probable.

The prognosis is influenced to some extent by the type of the disease. Abortive pneumonia is of short duration and favorable termination, as its name indicates. Pleuropneumonia is always a serious disease. There is in it a great liability to the development of empyema, or occasionally of chronic pneumonia. In infancy death is very liable to occur even early in the course of the pleuropneumonic attack. The nervous symptoms of cerebral pneumonia are not in themselves of bad prognostic import unless occurring late in the disease. Wandering pneumonia is more dangerous on account of the longer duration and necessarily greater exhaustion produced. The development of pneumonia in the course of other acute diseases adds naturally to the danger; and this is equally true of pneumonia attacking those already enfeebled by imperfect hygienic conditions or by any chronic ailment. Among other prognostic influences is the character of the epidemic, the malignancy of the disease being decidedly greater at some times than at others. The influence of age is very decided. The great majority of fatal cases are in subjects under 3 years, and especially under 1 year of age. The extent of the lesion bears some relationship to the prognosis; but only to a certain degree. Very widespread lesions give a more unfavorable prognosis.

In general the mortality figures of croupous pneumonia in early life are surprisingly low. As reviewed by Taylor[1] and by Schlesinger[2] the usual mortality in children, including infancy, according to a number of statistical calculations, is from 3 to 4 per cent., some series being decidedly over this. This is in sharp contrast to the high mortality for adults. The figures for the first 2 years of life, however, are distinctly higher, even reaching in some exceptional series approximately 25 per cent. in hospital practice. In private practice the mortality is lower. Uncomplicated pneumonia of an undoubtedly croupous nature in healthy subjects in childhood almost always recovers, and even in infancy the mortality is low. Without question many cases reported as fatal have been in reality instances of bronchopneumonia. A small number recover from the acute attack, but develop abscess or gangrene of the lung, interstitial pneumonia, or tuberculosis as sequels. A considerable proportion develop empyema. (See p. 82.)

Diagnosis.—This is usually easy in fairly typical cases but not by dependence on physical signs alone, especially in infancy. As already pointed out these signs are often late in appearing, or may be overlooked if the lesion is small. The diagnosis in general rests upon the sudden onset with a convulsion or chilliness and not infrequently vomiting; the rapid rise of temperature, its persistence at a high elevation, and its final rapid fall; the increased rate of respiration with the ordinary signs of usually moderate dyspnea; the possible slight cough; the suppressed cry or expiratory moan in infants; and in general the aspect of decided illness. The physical signs are those of pneumonia in general with the modifications already described, and are usually limited to one lung.

Owing, however, to the irregularity in symptoms and in the appearance and character of the physical signs, mistakes in diagnosis are easily made. The disease is to be differentiated especially from simple or tuberculous *bronchopneumonia.* The chief distinguishing features of bronchopneumonia consist in its frequent existence as a secondary disease; a slower and more insidious onset; slower development of physical signs, which are oftener in diffuse lesions with scattered mucous râles due to the attendant bronchitis; involvement generally of both lungs, especially the bases; slower rise of temperature and a more irregular temperature-curve with frequent remissions at uncertain times; decidedly greater degree of dyspnea; longer course, and more gradual disappearance of symptoms and physical signs. But frequent failure, on the one hand, of croupous pneumonia to run a typical course and, on the other hand, the not uncommon occurrence of pseudo-lobar forms of bronchopneumonia, as well as other deceptive symptoms, make the diagnosis often one of the greatest difficulty. Early in the attack the diagnosis between the two conditions is often impossible; and it may not be until it is over and one can view the completed picture that a conclusion can be reached. Finally there are repeatedly seen cases of bronchopneumonia the onset and course of which have been so similar to the most typical croupous form that only autopsy has permitted of a diagnosis. It is beyond question that many of the cases in infancy diagnosed as bronchopneumonia and recovering were in reality instances of croupous pneumonia; and conversely that many fatal instances of what was supposed to be the croupous form, autopsy would have shown to be bronchopneumonia.

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[1] Rep. Soc. Study of Dis. in Children (Brit.), 1907, VII, 203.

[2] *Loc. cit.*

The next most frequent cause of difficulty in diagnosis is the chance of the condition being *pleurisy.* Early in the attack the diagnosis is not always possible. The principal differential diagnostic marks of pleurisy in typical cases, after effusion has developed, are the slower development; greater degree and extent of dullness on percussion combined with feeble voice and breath sounds; absence of vocal fremitus; absence or feebleness of bronchitic râles; a percussion-dullness shifting with change of position; fullness of the intercostal spaces and increased resistance there on palpation; greater diminution in the respiratory expansion of the affected side; displacement of the apex beat in lesions of the left side of the chest; feebleness of the voice and breath-sounds even if these are of a bronchial character. The dullness is most intense in the lower part of the chest and posteriorly. Yet the determination of the nature of the disease cannot always be made with certainty. The bronchial character of the voice and of the breath-sounds in pleural effusion with compression of the lung are often very similar to those in pneumonia and the absence of tactile fremitus is not in early life a positive indication of any pathological condition. Frequently only the employment of exploratory puncture can settle the diagnosis, and this should always be made in doubtful cases. In pleuropneumonia the diagnosis is, of course, rendered doubly difficult. The high fever and other constitutional symptoms point to a pneumonia; the physical signs rather suggest pleurisy in the extreme dullness, increased resistance on percussion, and the diminution of vocal fremitus; but resemble pneumonia in the increased vocal resonance and the bronchial respiration. Exploratory puncture may give no positive results, owing to the exudate being largely plastic.

Delayed resolution in pneumonia is to be distinguished from a *slowly disappearing plastic or serous pleurisy* by the presence of decided auscultatory signs of consolidation with the absence of those of pleurisy; although the evidences from percussion may be equivocal. *Chronic* *interstitial pneumonia* may exhibit physical signs not readily distinguishable in some respects from those of delayed resolution; but differs in the history of the case, the character of the symptoms, the presence of evidences of contraction of the lung or of bronchiectasis, and in the fact that there is not, as in delayed resolution, a final disappearance of all symptoms and physical signs.

Cerebral pneumonia suggests often the possibility of the presence of *meningitis* of some kind, and early in its course the diagnosis may be impossible. Leucocytosis occurs in both conditions. The employment of lumbar puncture will be of service early in the attack; while later the development of the physical signs of pneumonia renders the diagnosis clear, except in those cases where pneumonia and meningitis are combined. Cerebral pneumonia exhibits its nervous symptoms usually at the beginning of the attack and these last, as a rule, but a short time. Meningitis occurring as a complication of pneumonia comes on later in the disease when physical signs of the pulmonary disorder are already present.

Grippe at its onset often resembles pneumonia very closely. There may be a convulsion followed by sudden, rapid rise of temperature, cough, and rapidity of respiration. Only after several days can pneumonia be excluded by the failure of characteristic symptoms and physical signs to appear. So like are the symptoms that it is only after the encountering of several such cases in the course of an epidemic of grippe that the physician hesitates to make the diagnosis of incipient pneumonia.

Scarlet fever is sometimes suspected on account of the initial vomiting and high fever. This is true also of cases of *gastrointestinal disturbance.* In some instances of pneumonia with few constitutional manifestations it is only the existence of a respiratory rate out of proportion to other symptoms which distinguishes the condition from what would otherwise be supposed to be an attack of indigestion with fever. A mistake of this sort is one frequently made. Appendicular pneumonia so closely resembles the onset of *appendicitis* that operations for this condition have repeatedly been needlessly performed. (See Appendicitis, Vol. I,p. 808.) The only safeguard is a careful, repeated examination of the lungs before operation is done.

The mistake of overlooking a pneumonia is much more common than that of wrongly considering it present. Consequently every child with sudden development of high temperature should have its chest carefully examined as a routine measure, and the examination repeated frequently as long as the diagnosis is not entirely beyond question; but in doing this it must never be forgotten, as already pointed out, that the diagnosis of pneumonia in early life rests more upon the symptoms than upon the physical signs.

Treatment. Prophylaxis.—Although only feebly contagious, yet the chance that it might be acquired from others with the disease must be borne in mind. Consequently children with infectious diseases or those in hospital wards with any affection of the respiratory apparatus are best kept separate from those suffering from pneumonia; and in family practice the well children should not for their own sakes sleep in the room with a pneumonic patient. Although this safeguarding is in theory certainly advisable and should be carried out whenever possible, yet the actual spread of the disease from one to another is not often encountered. Some of the family outbreaks of pneumonia which have been reported very probably were dependent upon a grippe infection. Precaution should be taken, too, against the occurrence of severe chilling of the body, and any catarrhal state of the respiratory mucous membrane should receive treatment promptly, to remove the susceptibility to the entrance of the pneumococcus into the lungs.

Treatment of the Attack.—Of the greatest importance here is to remember that croupous pneumonia in children is a self-limited disease which runs a short and usually a favorable course, and cannot be aborted by any specific treatment so far as yet known. Consequently the treatment is to be hygienic and symptomatic, and overdosing or overtreatment of any sort is to be avoided. The child should be kept in bed, and the windows of the room kept wide open, since there seems little doubt that the children feel better when given an abundance of fresh, cool air. If the weather is cold the child should be thoroughly wrapped in blankets including the arms, the head covered with a woolen cap, and hot-water bottles placed at the feet. It must be said, however, that although the cold-air treatment appears to make the child more comfortable, it is not proven that it has any influence upon the mortality of croupous pneumonia, which, as stated, is as a rule low in childhood (Morse and Hassman).[1] In the season of the year when the air is not cold the amount of covering on the bed will depend on the degree of fever, the temperature of the weather, and the surface-temperature and sensations of the patient. As a rule it should be light. There is no danger from the chilling of a patient with high fever, but care should be taken that the extremities are kept warm. Fever should be controlled by hydrotherapy, using cool sponging or warm tubbing or the cool pack, according to the age of the patient and the result obtained; and remembering that the mere reduction of the body-temperature is not the great desideratum. If the various nervous symptoms are improved by hydrotherapy the result is good. If the child resists greatly the treatment and is made more excited by it, while the pulse becomes weaker and more rapid and cyanosis develops and is slow in disappearing, the treatment should not be continued. Bodily and mental rest are what is to be sought. Water should be given frequently, and food, liquid or semi-liquid, at regular intervals; but the latter not insisted upon early in the attack if the child opposes it greatly, since at this time the appetite is often continuously absent. The child may take the position in bed most comfortable to it, but must not be allowed to lie indefinitely without change of this. If dyspnea is great the patient may feel easier when propped upon pillows. Applications to the chest are in my experience useless in modifying the course of the disease. When the crisis occurs an element of danger from collapse develops and the child must be kept warm by hot bottles, perhaps a hot mustard pack, and the administration of cardiac stimulants.

Treatment of the Special Symptoms.—Various of these may demand treatment in certain cases. *Pain* is often distressing. It may be relieved by opiates in moderate doses if there is no contraindication, such as tympanites, involvement of the respiratory centers, or a tendency to coma. In some instances the temporary application of an ice-bag, hot poultice, turpentine stupe, or mustard plaster to the side is of service. *Cough* is not usually annoying enough to demand medical treatment. Inhalations of warm water-vapor may be used; or, if frequent and causing loss of rest, a small dose of an opiate will relieve it, unless otherwise contraindicated. High *fever* is best treated by hydrotherapy, or, if this is not well tolerated, by antipyrine or phenacetin, 1 grain (0.065) or less at 2 years of age. This is, however, not often required. High temperature seldom demands treatment for itself, but for the *nervous symptoms* which often accompany it; or these may be present without any unusual elevation of temperature. Sleeplessness and nervousness are especially to be relieved. If hydrotherapy is not of service, they may be helped by antipyrine, phenacetin, the bromides, or an opiate. It is important in giving the coal-tar derivatives to employ small repeated doses rather than amounts which could produce too great a fall of temperature and consequent prostration. An ice-bag to the head may be of value but its action must be carefully watched in the cases of infants.

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[1] Amer. Jour. Dis. Child., 1916, XII, 445.

Cardiac weakness requires the administration of digitalis, strophanthus, or alcohol. While the employment of any such stimulants should not be followed as a routine measure, it is my belief that it is wise to give them on the first indication of cardiac debility, such as slight cyanosis or pallor, an unusual increase of the pulse-rate or respiration-rate, or a valve-like quality of the first sound of the heart, rather than to delay long. It is easier to maintain the cardiac strength than to revive it after it has failed decidedly. Nevertheless the majority of cases of croupous pneumonia do not require stimulation of any sort. A child of 2 years may receive from 1/4 to 1 dram (0.9 to 4) of whiskey or its equivalent every 3 hours according to the demands of the case. Camphor or caffeine sodio-benzoate given hypodermically is often of benefit as a cardiac stimulant especially in emergency, and nitroglycerine acts most favorably in many instances where cyanosis is present, by rendering the work of the heart easier. A dose of 1/200 to 1/100 of a grain (0.0003 to 0.0006) every 2 or 3 hours may be given to a child of 2 years. Epinephrine (1 : 1000 solution) in doses of from 2 to 4 minims (0.123 to 0.246) hypodermically is sometimes of value in emergency. Strychnine is excellent for continued administration as a cardiac and general tonic, but should not be pushed to the extent of increasing excitability. It is, in my opinion, often given too freely. The dose may be 1/200 of a grain (0.0003 or less every 3 hours at 2 years. (On the use of stimulants in pneumonia see also Bronchopneumonia, p. 65.) Carbonate of ammonia is, I think, of little value, and is a distinct gastric irritant. It is particularly at the crisis that energetic cardiac stimulation may be required. For cyanosis with severe dyspnea the inhalation of oxygen may be used with advantage. Atropine is of value in some instances where dyspnea is severe, especially if an attendant bronchitis increases the respiratory difficulty.

For the troublesome and often dangerous tympanites of severe cases gentle purgatives or the opening of the bowel by injection should be employed, or relief attempted by the insertion of a rectal tube, the application of turpentine stupes and the administration by mouth or by bowel of asafetida. In some instances eserine may be given hypodermically in doses of 1/500 to 1/300 of a grain (0.0001 to 0.0002) at 2 years of age. It is sometimes surprisingly successful, but is a powerful depressant drug and must be used with care. It is important to remember that expectorants are not indicated during the attack of croupous pneumonia. They can do no good and very readily disorder the digestion. The fibrinous exudate is not expectorated to any extent, but absorbed.

The importance of the matter of the therapeutic management of croupous pneumonia justifies the reiteration: That the best medicinal "treatment" of the average case of pneumonia is the absence of it.

There is a prevailing tendency to over-treatment in this disease. Skillful nursing and great watchfulness by the physician are the important matters, with a prompt but not officious readiness to help Nature when required.

Specific Treatment.—At various times different specific antipneumococcic sera and vaccines have been tried, but without satisfactory uncontested evidence that they exerted any influence upon the course of the attack. The obtaining by Cole[1] of a serum which seems to have a specific action upon the Type I of the pneumococcus is encouraging; but is of little service in the disease in early life, since the great majority of cases at that time do not depend upon this strain of the germ.

Convalescence is usually rapid and little if any treatment is required. The diet may be increased largely without delay if the appetite has returned. Should debility or anemia be present and persist, appropriate tonic measures are indicated. The child should be kept in bed until all evidence of consolidation has disappeared. This is, as a rule, only a matter of a few days. An exception may be made in the case of much delayed resolution with absence of symptoms; such children being cautiously taken out of bed regardless of the physical signs. The slow and irregular pulse-rate often seen after pneumonia is usually not a matter of moment, and does not make confinement to bed necessary.

CHRONIC PNEUMONIA

(Interstitial Pneumonia)

Etiology.—After some cases of croupous pneumonia there remains for weeks or even months a condition of consolidation, the lung failing to undergo resolution. I have known this to last for months, yet with final entire recovery. (See Delayed Resolution, p. 84.) It is not, however, to this that the title "chronic pneumonia" is properly applied; but to the long persistence of constitutional symptoms and physical signs, which in the large majority of cases of chronic pneumonia occur as a sequel to an attack of acute bronchopneumonia. Only in rare instances is the disease seen after the croupous form of the ordinary type; somewhat oftener after a pleuropneumonia. It may exceptionally occur, too, after chronic bronchitis, chronic passive congestion of the lungs, or chronic pleurisy; and it is likely that hereditary syphilis is a factor in some cases. Chronic interstitial pneumonia is especially liable to develop when there have been repeated attacks of the acute form, or when this has occurred in connection with the infectious diseases, chiefly measles, pertussis and grippe, or with the presence of a foreign body in a bronchus. It is sometimes tuberculous in nature, either primarily or by a later secondary infection. The disease is more common in childhood than later, and is most frequent in later infancy and the earlier part of early childhood; the period when bronchopneumonia is most often observed.

Pathological Anatomy.—Following a pneumonia which does not resolve, there is a thickening of the exudate in the alveolar walls, with a small-celled infiltration into the alveolar connective tissue, and soon a change of the infiltration into fully formed connective tissue, and an extension and subsequent contraction of this, producing shrinking of the lung. In fully developed cases there are bands of connective tissue passing in different directions through the lung, starting from the pleura, which is itself much thicker than normal. The alveoli are obliterated by organizing infiltration, their walls as well as those of the small bronchi are much thickened, and in many places these bronchi are dilated and filled with secretion.

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[1] Trans. Assoc. Amer. Phys., 1913, XXVIII, 606.

Macroscopically the lung in the affected region is found firmly adherent to the pleura, and only detachable with tearing. It is smaller and whiter than normal, contains less air, and on section cuts with a grating sound. The process is irregularly distributed, healthy areas being adjacent to those diseased, and the grey or white bands of connective tissue being distinctly visible. The lesions are most marked about the bronchi, and in advanced cases many cylindrical or saccular bronchiectases are observed. Usually only a part of one lobe is involved, although in severer cases there may be scattered lesions in different lobes or an entire lobe may exhibit the cirrhotic process. The lower part of the lung is more often attacked than the apex, except in tuberculous cases. In the most advanced areas which have lasted over a year no trace of healthy pulmonary tissue may be discoverable in the parts involved, and section show only much dilated bronchial tubes filled with muco-pus.

Symptoms.—After an attack of bronchopneumonia in which the fever and cough have been of unusually long continuance and the general condition severe, the symptoms may finally disappear except perhaps a certain degree of debility and anemia, with moderate cough and shortness of breath. Some traces of the physical signs of consolidation have, however, remained, and there may be even decided dullness on percussion, bronchial respiration, and the presence of râles. In still other cases the physical signs are first found after an attack of pertussis in which no acute bronchopneumonia had been discovered; or they develop in the course of a prolonged gastrointestinal disturbance; or in still others the initial disease was considered to be a plastic pleurisy only, any pulmonary involvement having been overlooked. Then after a variable time, perhaps some months, there may be a second attack of pneumonia with slow convalescence; or without this there may develop more or less fever of a remittent type, and there is a progressive loss of weight and strength; troublesome cough; increasing dyspnea; and sometimes pain in the chest, sweating, clubbing of the fingers, and diarrhea. Fever may be absent for days and return again and reach possibly 104° or 105°F. (40° or 40.6°C.).

As the disease advances the physical signs become characteristic. Evidences of decided shrinking of the lung appear. If this is at the apex there will be depression of the supraclavicular fossa. If in the region of the heart an abnormally large cardiac dullness and unusually distinct pulsation are observed. There are also decided dullness on percussion, diminished expansion of the affected region, and very commonly a peculiarly feeble respiratory murmur. Sometimes bronchial respiration and bronchophony are found, if not present earlier; and over large bronchiectases there may be a tympanitic note on percussion and a respiratory murmur suggesting the existence of a cavity. The diaphragm may be drawn upward on the affected side, and eventually there may develop very decided deformity of the chest. The intensity of all the signs depends upon the extent and degree of pulmonary change which has taken place. They may vary, too, from day to day. If only a partial consolidation exists, the respiration may be merely bronchovesicular or enfeebled, with slight impairment of percussion resonance. Bronchitic râles are a variable element, present only at such times as an attack of bronchitis coexists. When bronchiectasis is well-developed and has become a prominent lesion, there is usually abundant mucopurulent secretion of a more or less offensive odor. Hemoptysis sometimes occurs.

Course and Prognosis.—Until decided shrinking of the lung through the interstitial contraction has taken place, complete resorption with disappearance of the physical signs is possible, yet it may take months for this to be accomplished. After this shrinking has occurred the passing of the physical signs is impossible; but this by no means precludes recovery from the majority of the symptoms. In most cases the course of the disease is typically chronic, with a tendency to gradual involvement of a whole lung, perhaps after the elapse of several years; with return of the symptoms, or exacerbations of them from time to time, shown by bronchitis, fever and increased cough and dyspnea. The general health gradually suffers and the patient is always more or less delicate, and finally death occurs after months or years of increasing emaciation, anemia and exhaustion. Others die of a recurrent attack of pneumonia. Many of the cases, if not tuberculous at the onset, finally develop this disease.

Diagnosis.—This rests chiefly upon the history of the case and on the presence of persistent physical signs of consolidation after a pneumonia. *Delayed resolution* of a pneumonia is to be distinguished by its association with the croupous form of the disease, the absence of the symptoms of chronic pneumonia as described, and the final recovery after a much shorter time than in chronic pneumonia. Delayed resolution in bronchopneumonia is characteristic of the protracted form of that disease (p. 84), and is in many cases but the beginning of a chronic pneumonia. *Pleural effusion* is to be excluded by its more widespread and absolute dullness on percussion, the presence of aegophony, and the absence of vocal fremitus. The last sign is, however, equivocal, since the thickening of the pleura seen in chronic pneumonia may readily diminish vocal fremitus, and the sign is in any event difficult to elicit in children. The employment of the exploring needle is the surest diagnostic method between the two conditions, but is not without some danger if the disease is an interstitial pneumonia. The most important and the most difficult diagnostic question is between the *simple* and the *tuberculous* forms of chronic pneumonia, and this frequently cannot be satisfactorily determined. Tuberculosis affects more often the apex; simple chronic pneumonia the base. The cutaneous tuberculin reaction, if negative, indicates the simple form of the disease; if positive no certain conclusion can be drawn. Repeated examination of the sputum is the most serviceable diagnostic method, although the finding of bacilli does not prove that the affection was primarily tuberculous, and not a chronic pneumonia with a later secondary tuberculous involvement.

Treatment.—This is essentially tonic in character. The administration of cod-liver oil is one of the best methods, and iodide of potassium is to be recommended in some cases. The clothing and the habit of life in general should be such that exacerbations of the disease are prevented. Cool morning baths are of service if well tolerated. Abundant digestible nourishment is necessary. Change of climate and life in the open air are among the best of remedies to check further progress of the disease, if it has passed the possibility of cure. Pulmonary gymnastics are of service in expanding the diseased lung. When there is decided bronchiectasis the treatment recommended for this condition is to be employed (p. 46).

HYPOSTATIC PNEUMONIA

This is not a primary disease, but the result of prolonged passive congestion from various causes. It may attend, for instance, severe typhoid fever, or occur in any marantic state such as seen in the chronic gastroenteric diseases of infancy. Lying long in one position may favor its development. The **lesions** consist of a low-grade inflammation of the pulmonary tissue beginning in the lower portion of the lung. This becomes overfilled with fluid and the blood-vessels congested, while the alveoli contain an exudate of desquamated and partially degenerated epithelium, red blood-cells, and leucocytes. The lesions differ from mere congestion in the presence of this exudate. The pleura is not involved. The process is lobular in nature and affects only the more dependent portions of the lung, generally on both sides and in both the upper and lower lobes, being thus widespread but near the posterior surface. In appearance the tissue is dark red in color, airless, and firm to the touch. There is no attendant pleurisy.

The **symptoms** are uncharacteristic and the condition is usually recognized only at autopsy. There is neither dyspnea nor fever, unless dependent upon the primary disorder. The physical signs consist principally in slight dullness on percussion, feeble respiratory murmur, and the presence of moist râles. In the way of **prognosis** the condition is of no importance, except as a sign of the influence which the primary disease is exerting. Hypostatic pneumonia is not in itself the real cause of death, although it increases the danger.

The **treatment** is preventive, consisting in that of the primary affection, in the sustaining the general and circulatory strength, and in frequent change of position.

PULMONARY EMPHYSEMA

Etiology.—Emphysema, always a secondary affection, may be either *acute* or *chronic*. The former is very common and is seen oftener in childhood than at any other period. It may be produced in the course of any affection where the air-pressure within the lungs is increased. It is present to a certain extent in all acute pulmonary diseases in children, and may be a *compensatory* process in pneumonia, atelectasis, or where pleuritic adhesions interfere with the expansion of some portion of the lung. In other cases the emphysema is *obstructive* and follows forcible coughing such as occurs in bronchitis and pertussis, or is the result of obstruction to respiration as in diphtheria of the larynx. The more chronic form is less common in early life, and is seen then chiefly in later childhood. It is liable to follow conditions of long-continued dyspnea, as in rachitic deformities, tuberculosis, chronic pneumonia, asthma, chronic bronchitis, adenoid growths, and tracheobronchial adenitis, or it may persist after severe acute bronchopneumonia or pertussis.

Pathological Anatomy.—In *acute vesicular emphysema* there is merely a distention of the vesicles, with but little rupture of these and consequent entrance of air into the parenchyma. It may be generalized, but is more frequently limited to certain regions, oftenest the anterior border of the lungs especially of the upper lobe, or may be scattered among or surrounded by areas of consolidation or of atelectasis, if these are present. The affected portion is paler and more prominent than normal and does not collapse readily. The contrast between areas of this sort and healthy lung is striking, and is still greater between such areas and any neighboring consolidated or atelectatic portions. When much of the anterior border of the lungs is emphysematous the heart may be completely covered. In *chronic emphysema*, which also may be generalized or partial, the dilatation is permanent owing to loss of elasticity, and many of the alveolar septa are ruptured, the alveoli communicating with each other and producing distended sacules of pin-head up to pea-size or larger. The capillaries are more or less obliterated. As a further result of the rupture the air may enter into the interstitial pulmonary connective tissue (*interstitial emphysema*), or accumulate between the lobules (*interlobular emphysema*) and compress the alveoli, or even under the pleural surface or about the root of the lung or in the mediastinum, from which situation it may find its way into the subcutaneous connective tissue (*subcutaneous emphysema*). (See p. 540.)

Symptoms.—These are not definite, being masked by those of the attendant primary disease. The physical examination shows marked hyperresonance on percussion, perhaps with obliteration or diminution of the cardiac dullness and depression of the upper border of the hepatic dullness, and accompanied by feeble respiratory murmur and diminished vocal fremitus. In the acute cases the symptoms disappear sooner or later as recovery from the primary disease takes place. In those of a chronic nature the alteration of the percussion resonance is persistent; the chest may assume the barrel-shape characteristic of emphysema in the adult, and the expiratory excursion is limited. This is especially true of the emphysema accompanying asthma. The presence of emphysema adds to any dyspnea and cyanosis which the primary disease may produce. The only certain clinical evidence of the interstitial form is the rare occurrence of subcutaneous emphysema, developing first in the neck. This condition is oftenest secondary to interstitial emphysema developing at the root of the lung.

The **prognosis** depends entirely upon the primary disease. Emphysema may add to the severity and danger of this, but is not in itself a cause of death. In the cases attending acute disease the lesion is transitory and disappears largely or completely in the course of a few weeks.

The **treatment** is entirely that of the primary affection, and there are no special measures indicated.

ATELECTASIS

Atelectasis has already been considered under the Diseases of the Newborn. Here only we shall discuss the disease as it is encountered at periods later than the first few weeks of life.

Etiology.—One of the principal causes of atelectasis in infancy is unusually well-developed rachitis. The deformity of the chest, the lowered resiliency of its walls, and the diminished muscular power readily favor the development of more or less collapse. Other deformities, as from caries of the spine, also predispose to it, as does the existence of any debilitating disease. Consequently it is a frequent condition in all atrophic or debilitated states in infancy, the tendency to it being increased by the much weakened force of the cough at this period and the feeble respiratory power. It is produced also by the pressure of intrathoracic tumors, pericardial effusion, pneumothorax and especially pleural effusion. A very common cause is severe bronchitis, in which collapse of the vesicles follows obstruction of a bronchus by mucus; and it is present more or less in most cases of bronchopneumonia. Acquired atelectasis is thus an attendant upon numerous other affections, but does not exist as a primary disease.

Pathological Anatomy.—The lesions are similar to those previously described in considering Congenital Atelectasis (Vol. I, p. 284), except that in the acquired form they are usually not so extensive, and that the position is more variable, depending upon the cause. Certain regions become airless, congested, deep-red or purple in color, hard, and depressed below the neighboring paler, healthy or usually emphysematous lung. The distribution of the atelectasis is lobular, and these lobules may be scattered throughout the lung; while in other cases many such areas may be aggregated, until a considerable portion of a lobe may become atelectatic. The favorite seat is the lower lobe or the posterior portion of the upper lobe. When the atelectasis depends upon local compression, as by a pericardial effusion, the region involved corresponds with this. In cases of long duration connective-tissue changes take place and the lung shrinks. This may occur in cases of compression-atelectasis attending empyema.

Symptoms.—The symptoms and physical signs are much the same as in the congenital form. The onset is often gradual; there are rapid, shallow respiration; dyspnea with inspiratory recession of the thoracic wall; rapid feeble pulse; normal or subnormal temperature, and cyanosis. On physical examination of the chest a feeble respiratory murmur with moist râles is found and perhaps slight dullness on percussion. Yet both symptoms and physical signs are quite commonly masked by those of the primary disease, and the atelectasis may be discovered only at autopsy. It is indeed surprising how few symptoms a child with decided chronic atelectasis may exhibit. In other cases the disease often cannot be distinguished clinically from hypostatic pneumonia, with which, or with bronchopneumonia, it is liable to be associated.

Prognosis.—This depends upon the cause, as well as upon the age. Cases due to pleural effusion, even when the collapse is comp ete, recover entirely unless adhesions or overgrowth of connective tissue prevent reexpansion. In severe rickets, on the other hand, the prognosis is unfavorable, because the condition of the chest-walls is slow in altering for the better, if, indeed, it does so at all. In all chronic debilitated conditions, and in disorders obstructing respiration, the prognosis of atelectasis is unfavorable, especially under the age of 2 years, and the collapse of the lungs increases greatly the danger already existing.

Treatment.—In the line of treatment is that described for congenital cases; consisting of frequent change of position, stimulating warm or hot baths, hot mustard-packs, maintaining the bodily temperature, fresh air, inhalations of oxygen, and the like.

EDEMA OF THE LUNGS

This is a symptom, not common in childhood, which may be seen in various disorders, principally to be mentioned here being valvular disease of the heart, rachitis, nephritis, bronchopneumonia, eruptive fevers, and cachectic conditions. The lungs on post-mortem examination are firm, heavy, red or grey in color, and crepitate little if at all, but do not sink in water. On section an abundant watery, frothy fluid exudes freely. The lesions of atelectasis, congestion, or pneumonia may also be present

The symptoms are not characteristic and the condition is generally recognized only at autopsy. Suggestive, especially when there are evidences of effusion of fluid elsewhere in the body, or if nephritis exists, are increase of percussion dullness in the lower portion of the chest posteriorly without bronchial respiration; the presence of very numerous moist râles; and the existence of cyanosis, cough and dyspnea.

From the point of view of prognosis pulmonary edema is an unfavorable symptom. When developing suddenly in cases of nephritis death is liable to occur promptly. Under other circumstances it is an indication of the severity of the diseased condition on which the edema depends, and is often the sign of danger, but usually not in itself the cause of it.

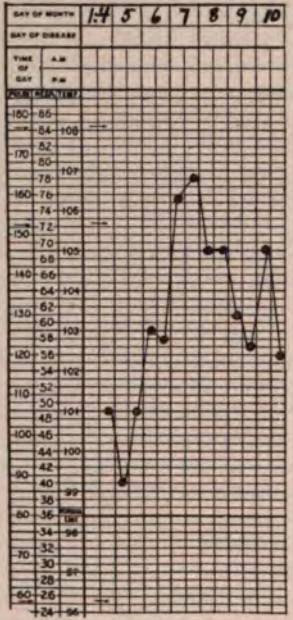

Fig. 295.—Multiple Pyemic Abscesses of the Lungs Following Cervical Adenitis.

Rose T., aged 5 years. Illness began a week before admitted to the Children's Hospital of Philadelphia, on Jan 4, with inflammation of the tonsils and of the cervical gland on the left side; Jan. 5, incision of abscess in neck; Jan. 7, child toxic, delirious, general condition poor; Jan. 10, numerous râles have been present in both lungs. No other positive physical signs, except a slight bronchial respiration in the lower part of right side. General condition grew worse. Death on this date. *Autopsy* showed numerous scattered pyemic abscesses, size of walnut, throughout both lungs. Pus in pleural cavity on both sides.

Treatment is required only in the rapid, severe cases. In these dry cupping, free cardiac stimulation, purgatives, and hot mustard-packs are to be used.

PULMONARY ABSCESS

Etiology.—Small multiple pulmonary abscesses may attend sepsis (Fig. 295) or severe bronchopneumonia, or larger ones may be seen in tuberculosis. A non-tuberculous abscess of considerable size is of very exceptional occurrence in early life. It is to this condition that reference is especially made in this connection. It may be the result of the entrance of foreign bodies, or of a disintegrating consolidation in croupous pneumonia, or may sometimes be secondary to a suppurating bronchial gland. In 5767 cases of disease in children Bullet[1] reported 10 instances of abscess of the lung, 7 of these being in subjects of from 1 to 3 years of age. Other cases have since been reported from time to time. I can recall but 3 instances under my own observation, the diagnosis of which appeared to be beyond question.

Symptoms.—The symptoms are variable and uncharacteristic. In some instances the condition is not suspected unless there chance to be a sudden rupture of the abscess into a bronchus and a discharge of pus containing elastic fibres, and sometimes possessing an offensive odor. Generally no such sudden rupture is observed, although communication with a bronchus is not infrequent. The principal clinical features consist of fever of an irregular and often hectic type, with increasing debility and anemia, a high leucocyte count, and at times a troublesome cough. When the abscess is large and there is free communication with a bronchus, a tuberculous or a large bronchiectatic cavity may be suspected. If no such communication is present, there is persistent dullness on percussion and feeble respiratory murmur over the consolidation, and a localized empyema seems probable. In cases of distinct localization the exploratory needle may reveal the presence of pus; but is very liable to fail to do so, owing to the smallness of the accumulation in size. Even when pus is found, the diagnosis of localized empyema is often erroneously made. Small, deep-seated abscesses may present no physical signs whatever. A very characteristic symptom of abscess, although not pathognomic, is decided clubbing of the fingers. This peculiarity was well-marked in a case of a pulmonary abscess in a child, due to the entrance of a watermelon seed into a bronchus.

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[1] Ref., Brandenberg, Jahrb. f. Kinderh., 1908, LXVII, 458.

Course and Prognosis.—The prognosis is always uncertain. The abscess may rupture into a bronchus, as already stated, and perhaps be followed by healing. This is especially true if the original cause was a foreign body which had been expelled by cough. In other cases the abscess may continue to discharge, with progressive loss of strength by the patient and the usual symptoms which prolonged suppuration produce.

If the **diagnosis** can be made surgical **treatment** should be employed. Aspiration may be followed by cure, but oftener incision, resection of a rib and drainage are required.

PULMONARY GANGRENE

Etiology.—This is not a common condition in early life and is always secondary. It is oftenest seen in connection with bronchopneumonia especially after measles; croupous pneumonia; tuberculosis of the lungs and bronchial glands; typhoid fever and other acute infectious diseases; aspiration-pneumonia, as of foreign bodies or especially of necrotic or gangrenous tissue in the case of diphtheritic or gangrenous stomatitis; septic embolism or thrombosis of the pulmonary vessels resulting from suppurative processes elsewhere in the body; and gastroenteritis. Any greatly debilitating agent predisposes to it. As a result of some of these causes inflammation and consequent interference with the circulation in the lung take place; but in order to have a gangrenous process follow, there must be the presence of microörganisms capable of producing putrefaction. These may enter the lung either by way of the respiratory tract or through the blood-vessels. According to the investigations of Veillon and Zuber,[1] these germs are anaërobic putrefactive bacteria of various sorts and appear to be specific for gangrenous processes.

Pathological Anatomy.—The lesions may be either diffuse or circumscribed; the latter being much the more frequent in early life. They are usually multiple and sometimes are present in both lungs but oftener in the right only, are of variable size, and situated oftenest in the lower lobe and near the surface. The affected region is blackish or greenish in color, moist, and at first harder than normal pulmonary tissue. Later it either still contains a slough or exhibits a cavity filled with pus. The odor is very offensive and characteristic. In the rarer cases of diffuse gangrene almost the whole of one lung or a large part of a lobe is heavy, moist, dark-greenish or black in color, and with a gangrenous odor.

Symptoms.—In well-developed characteristic cases there is a horribly putrefactive odor to the breath and to the expectoration, the latter consisting of dirty, greenish material, containing gangrenous pulmonary tissue and sometimes blood in large amount. The patient shows great pallor, high remittent fever, rapid feeble pulse, and profound prostration

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[1] Arch. de méd. experim. et d'anatom. path., 1898, X, 517.

(Fig. 296). There may be but little cough. The symptoms, however, are by no means always typical in children and the diagnosis may be made only at autopsy. Odor to the breath is probably oftener absent than present in early life. The physical signs are not characteristic. They suggest those of a lobular pneumonia or of a cavity according to the case; or of a pyothorax or pneumothorax if the gangrene has extended to the pleura and has perforated this.

Course and Prognosis.—The course is usually rapidly fatal in from 1 to 3 weeks. Gangrene due to the presence of a foreign body offers perhaps the best hope; but even this is slight. A few cases of recovery are on record (Renault).[1]

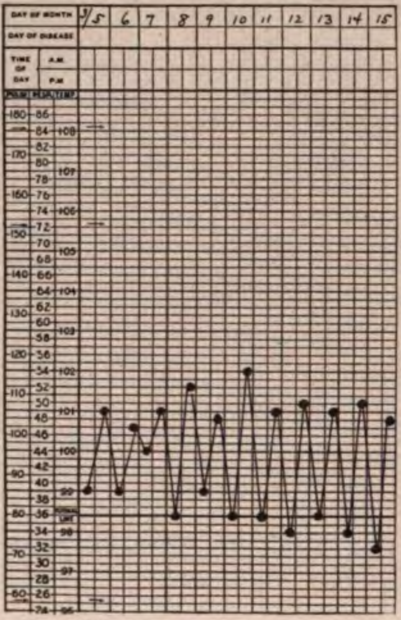

FIG. 296.—GANGRENE OF THE LUNG.

James S., aged 8 years. Seen Mar. 4, had grippe 11 weeks previously, followed by pneumonia. Signs of consolidation said to have cleared rapidly. About 4–5 weeks before seen developed daily fever, failing strength, violent paroxysms of coughing, with purulent expectoration and sometimes vomiting. Examination showed numerous râles at the lower part of right side posteriorly. Excessive weakness prevented further study. Odor of breath and of expectoration horribly offensive. Death Mar. 21.

Diagnosis.—This is necessarily usually difficult owing to the frequent absence in children of the characteristic symptoms. Even if an odor to the breath is present the possibility of fetid bronchitis, bronchiectasis, pulmonary abscess, or of gangrenous or ulcerative stomatitis must be taken into account. Only by excluding these and the finding of necrotic pulmonary tissue in the expectoration will the diagnosis be made sure.

Treatment.—Strongly stimulating and supporting treatment is indicated. Efforts should be made at pulmonary disinfection by the inhalation of antiseptic substances such as thymol, creosote, turpentine and eucalyptus. Operation is indicated if the gangrene is localized and situated near the surface of the lung.

[1] Grancher, etc., Traité des mal. de l'enf., 1904, III, 513.

CHAPTER VI

DISEASES OF THE PLEURA

PLEURISY

Although pleurisy is a common disorder in early life, it occurs nearly always as a secondary affection. It is probably only in later childhood that the primary form may be seen.

The disease may be divided into: (1) *The dry or plastic form,* (2) *the serous or serofibrinous form,* and (3) *the purulent form, or empyema.* These shade one into another to a certain extent; that is to say some cases of pleurisy which are at first plastic may produce later more or less serous exudate, while some effusions at first shown by aspiration to be, at least apparently, serofibrinous in nature, later become distinctly purulent.

Etiology.—The influence of *age* is very decided. In the newborn pleurisy is usually considered uncommon, at least in a clinically recognizable form. Steele[1] was able to collect but 25 cases including 1 of his own, and in addition to a few others previously reported by Hervieux.[2] The subject has been studied also by Roger,[3] Macé[4] and others. It appears oftenest to be purulent, frequently bilateral, and always secondary. After this period a moderate degree of plastic pleurisy is of frequent occurrence in both infants and children; and a purulent effusion, too, is relatively much more frequent than in adult life; up to the age of 5 years the majority of pleural effusions being of this nature. In 54 consecutive cases of pleural effusion in children under 2 years of age occurring in the Children's Hospital of Philadelphia, 51; *i.e.* 94 per cent., were purulent (Miller).[5] This preponderance of purulent pleurisy would appear to apply, however, only to clinically recognizable cases, since Reano[6] found 78 cases of pleurisy in 756 autopsies upon infants, and in 45 of these there was merely a simple deposit of fibrin.

According to Netter[7] $\frac{1}{3}$ to $\frac{1}{2}$ of the cases of pleurisy in infancy and childhood are purulent while in adults about $\frac{1}{16}$ to $\frac{1}{15}$ are so. In 642 cases of purulent pleurisy in children collected by him 62 per cent. were less than 5 years of age, and 27.6 per cent. were from 5 to 10 years. Serous pleurisy with large effusion, on the other hand, is a most exceptional occurrence in infancy and is relatively much less frequent at any period of early life than it is in adults. In later childhood it generally exceeds purulent effusion in frequency, the ratio from 5 to 10 years being about 6 to 1 according to Israel.[8] Considering all forms of pleurisy collectively with regard to age, this writer in 206 cases found from birth to 5 years, 45.6 per cent.; (5.34 per cent. being less than 1 year); from 5 to 10 years, 34.4 per cent.; from 10 to 15 years, 20 per cent.

Pleurisy occurs rather oftener in boys than in girls and the greater number of cases develop in the colder season of the year. Trauma is an uncommon cause in early life. Exposure to cold is a factor in later childhood, as is the existence of rheumatism. The various infectious diseases are often complicated by pleurisy. Sepsis in the new born may produce it; scarlet fever is a frequent cause; and measles, grippe, and typhoid fever exhibit it not infrequently. Tuberculosis often has pleurisy as one of its lesions, generally consecutive to involvement of the lung or of the bronchial glands. With this origin it is oftener serofibrinous than purulent. In some instances purulent pleurisy develops insidiously without any previous known illness to which it can be attributed; while serofibrinous effusion, particularly in later childhood, is apparently oftenest a primary affection, at least so far as any discoverable cause is concerned. How many of these "primary" cases are in reality tuberculous is a matter much discussed and still undetermined. By all means the most common factor in causing the disease is an extension from a pneumonic process. Probably nearly every case of pneumonia in early life is attended by a certain amount of plastic pleurisy, and the majority of cases of empyema in children owe their origin to pneumonia. Dunlop[1] found this true of 69 per cent. of 98 cases of empyema, and others give a higher figure. Either form of pneumonia may produce it, but the croupous variety is much the more frequent cause.

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[1] Phila. Med. Jour., 1898, Sept. 17.
[2] Journ. f. Kinderkr., 1864, XI, 371.
[3] Thèse de Paris. 1903.
[4] L'Obstetrique, 1900, V, 7.
[5] Arch. of Ped., 1911, XXVIII, 28.
[6] La Pediatria, 1913, XXI, 588.
[7] Grancher: Traité des mal. de l'enf., 1904, III, 645.
[8] Thèse de Copenhagen, 1882. Ref., Netter, *loc. cit.*, 641.

Bacteriology.—Germs of various sorts appear to be the exciting agent, although in many cases the number found is so small that the etiological relationship is uncertain. In the plastic and serofibrinous forms the pneumococcus is the germ oftenest present; less often the staphylococcus, tubercle bacillus or streptococcus; or no germs at all may be discoverable in many instances. It may be only by inoculation-tests that a serous fluid apparently sterile is shown to be in reality tuberculous. In purulent effusion likewise the pneumococcus is the germ observed by far the most frequently, either alone or associated with other species; less often the streptococcus is discovered in pure culture. Various other bacteria may sometimes be the active agents, among them the staphylococcus, colon bacillus, influenza bacillus, typhoid bacillus, etc. Netter[2] found the pneumococcus present, alone or associated, in 74.89 per cent. of 171 cases of purulent pleurisy examined; the streptococcus in 16.90 per cent. and the tubercle bacillus in only 9.84 per cent. Dunlop[3] in 98 cases discovered the pneumococcus in 71.42 per cent., oftenest in pure culture; the streptococcus in 32.6 per cent., staphylococcus in 6.12 per cent., and the tubercle bacillus in only 3.06 per cent. Similarly in 212 cases of empyema Koplik[4] observed the pneumococcus in 75 per cent. This is in sharp contrast to the condition in adults, in whom the streptococcus and the tubercle bacillus are present very frequently in pleural exudates. The cases of purulent pleurisy following pneumonia show oftenest the pneumococcus; those seen as one of the evidences of sepsis are oftener associated with the streptococcus or staphylococcus. The streptococcus alone or in combination with the pneumococcus is liable to be present in cases developing after scarlet fever, measles, and other infectious diseases.

The lesions, symptoms, and treatment of pleurisy depend so greatly on the form of the disease present that these forms must be considered separately.

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[1] Edin. Med. Jour., 1914, XIII, 4.
[2] *Loc. cit.*, 648, 649.
[3] *Loc. cit.*
[4] Diseases of Infancy and Childhood, 3d edit., 654.

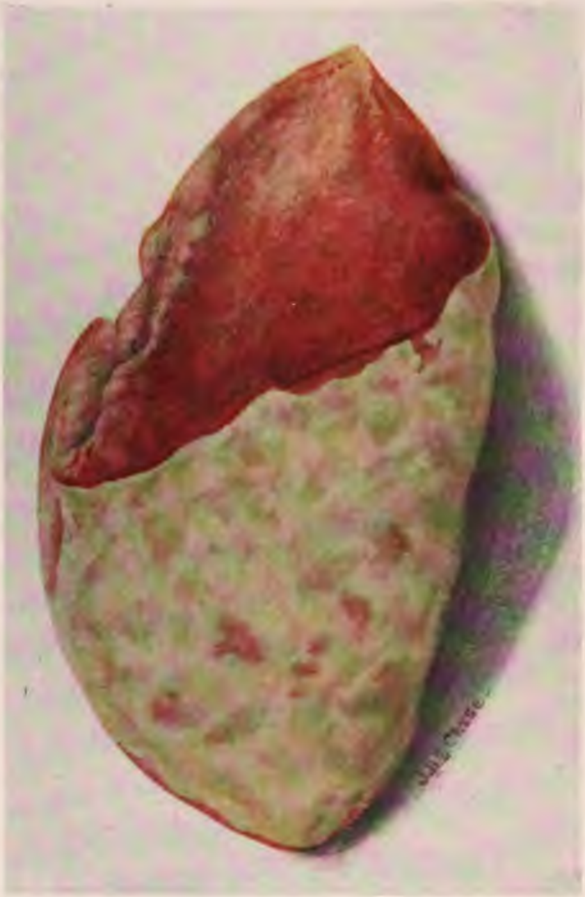

FIG. 297.—PLASTIC PLEURISY WITH PNEUMONIA.

Child of 16 months with symptoms of pleuro-pneumonia. Illustration shows the dense layer of plastic exudate found at autopsy. About 2 ounces (59) of yellow serous fluid in the pleural cavity.

1. PLASTIC PLEURISY

Pathological Anatomy.—In the affected region the pleura is rough, dry, injected and lustreless, and in many advanced cases covered with a distinct deposit of fibrin. This may exist only in shreds, or may form a thick layer binding the costal and pulmonary pleural surfaces more or less loosely together. The fibrin is readily detachable in recent cases; but if the condition has lasted some time the adhesions are firm and the fibrinous material cannot be easily removed. In color it varies from grey to greenish-yellow, depending upon the number of pus-cells included in its meshes. The condition is especially marked in well-developed cases of pleuropneumonia (Fig. 297). Pneumonic involvement of the lung is in fact nearly always present, except toward the end of later childhood, since the majority of the cases of pleurisy are secondary to or coincident with this disease. In the milder cases only the pleura covering the affected lung is involved, but in others the process is more extensive and the parietal pleura is also attacked. As recovery takes place the exudate is absorbed, but adhesions between the pleural surfaces usually remain. When the pleurisy is tuberculous in nature, grey and yellow tubercles may sometimes be seen on the pleural surface and in the fibrinous exudate, and the pleura becomes much thickened.

Symptoms.—These are very indefinite and usually overlooked, since the symptoms of the primary disease conceal them. Pain is the predominant manifestation, especially noted in taking a deep breath or on coughing; but infants cannot complain of this, and young children often do not. It is only in later childhood that this distinct evidence of dry pleurisy becomes manifest. The cough is then short, frequent and harassing; there is often more or less fever but sometimes none; and there may be tenderness on pressure. The pain is felt over the affected region or may be referred to the abdomen. The physical examination shows the presence of a pleuritic friction rub, which does not disappear on coughing or crying, and is heard both with expiration and inspiration. Sometimes the rub closely simulates the crepitant râle of pneumonia, but it is usually much more superficial in character.

Course and Prognosis.—The prognosis is good if the pleurisy does not pass beyond the plastic stage. The disease continues a few days or sometimes several weeks. It is only exceptionally in children that dry pleurisy runs a very chronic course, with great thickening of the pleura from organized connective tissue, and with obliteration of more or less of the pleural cavity. Such cases are likely to be tuberculous in nature. There is in many individuals a decided tendency to recurrence shown.

Treatment.—Apart from the treatment of the primary disease measures should be taken to relieve pain. An ice-bag to the affected region often gives relief, or phenacetin may be employed, or small doses of opiates given if needed. For the control of the inflammation counter-irritation is indicated, employing mustard-plasters in young subjects, or diluted tincture of iodine in older ones. The patient should be put to bed if not already there. A bandage around the chest restrains its movement and aids in preventing pain. The salicylates are believed to be of service in cases considered rheumatic, and often even in others. They do, it is true, relieve pain and possibly affect the course of the disease, but they are very liable to disorder the digestion.

2. SEROFIBRINOUS PLEURISY

Pathological Anatomy.—The early stages of this condition are the same as in plastic pleurisy. Soon, however, an exudate of serum from the blood-vessels takes place. This may be situated in small pockets of an extensive, thick plastic exudate and be in small amount; in other cases in larger pockets, shut off from each other by adhesions; in still others free in the pleural cavity, abundant, and possibly filling the whole of one side. Serous pleurisy occurring upon both sides is of rare occurrence. Barthez and Sanné[1] found it in 22 of 343 cases. In some cases the fluid far overbalances the plastic inflammation, and may be produced from the onset of the disease. In others it is secondary in development and in limited amount. The characteristics of the fluid, too, vary with the case, being nearly clear or quite turbid, depending upon the amount of fibrin and leucocytes contained in it. Small shreds or larger masses of fibrin may be found floating in the effusion. The wall of the pleura itself may be but little involved, or may show a connective-tissue hyperplasia and cellular infiltration. Very clear fluid is suggestive of a tuberculous origin, and the cells present in that case

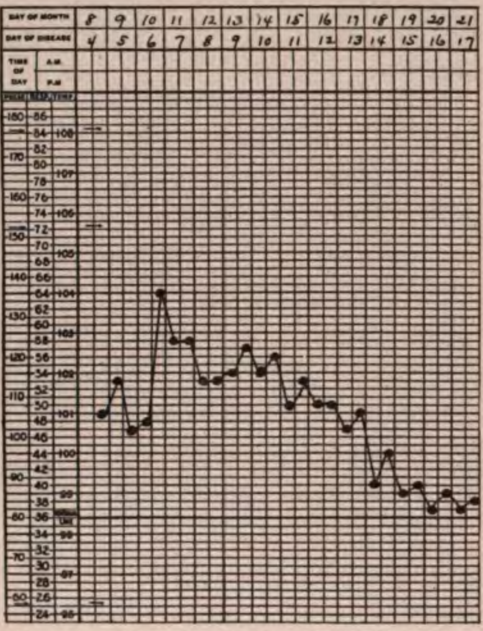

FIG. 298.—SEROUS PLEURISY, PROBABLY SECONDARY TO PNEUMONIA. ACUTE COURSE.

James N., aged 5 years. Entered Children's Hospital Dec. 8, with cough, fever, and pain in the left side of the chest. Said to have been ill since Dec. 5. Examination showed good general condition, dulness on percussion and bronchovesicular respiration with fine râles over lower part of the right side; Dec. 9, blood showed 15,000 leucocytes, 71 per cent. polymorphonuclears; Dec. 10, evidence of fluid now more marked, the dulness increasing and the respiration more distant, von Pirquet reaction positive; Dec. 19, general condition good, fever gone, but patient quite dyspneic, aspiration done, gave clear yellow fluid, no germs found, negative on culture; Dec. 28, recovery complete, except for slightly distant breath-sounds still remaining.

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[1] Mal. des enfants, 1884, I, 818.

are predominatingly lymphocytes. The fluid in serofibrinous pleurisy is usually gradually absorbed, but adhesions persist to a varying extent, and in some instances the pleura remains permanently much thickened. In others a fluid, at first clear or slightly turbid, rapidly changes into one of a purulent character. It is probable, however, that cases of this nature are not true instances of serofibrinous pleurisy, since the fluid from the beginning usually contains very large numbers of bacteria. An exudate of red blood-corpuscles in cases of severe inflammation may produce a *hemorrhagic exudate*, but this is uncommon in early life, Israel[1] finding it in only 2 instances in 206 cases. Lewin[2] discovered it rather more frequently than have others (4 in 50 cases of effusion). Hemorrhagic effusions are oftenest associated with rheumatism, tuberculosis, or malignant neoplasms.

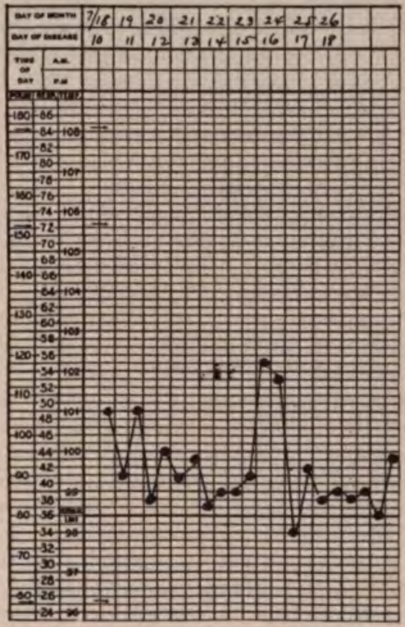

FIG. 299.—SEROUS PLEURISY, SOMEWHAT SUBACUTE FORM.

Robert McC., aged 10 years. Previous health been only fair. Attack began 10 days before with fever, pain in the abdomen, and headache. Examination by a physician at the onset gave negative results. Symptoms continued, and a week later signs of fluid were found. Has had no cough. *Examination* on admission to Children's Ward of the University Hospital, July 18, showed flatness over the left side nearly throughout, absent voice-sounds and breath-sounds, heart displaced to the right, moderate dyspnea. Aspiration next day withdrew 34 ounces (1005.5 c.c.) clear, straw-colored fluid. Dyspnea was relieved and did not return.

Symptoms.—The manifestations of the primary disease usually at first conceal those of the pleural effusion entirely, or continuously so where the effusion is small, as in many cases of pneumonia. In older children with what appears to be a primary serous pleurisy of considerable size there is a sudden rise of temperature, perhaps vomiting, dry hacking cough, and shallow and painful respiration as observed in plastic pleurisy. The child does not, however, exhibit the constitutional impression of disease to the extent characteristic of pneumonia. Fluid is now more or less rapidly effused, and sometimes in a very few days, usually later, one side of the chest may be nearly filled. Pain generally becomes less and often cough also, and the fever, which had ranged from 101° to 104°F. (38.3° to 40°C.) diminishes slowly (Figs. 298 and 299), or even disappears; but dyspnea increases. The degree of this varies with the amount of fluid. With regard to pain, it is to be noted that this may be chiefly abdominal and the suspicion of an appendicitis be aroused, analogous to the condition often seen in pneumonia. (See Appendicular Pneumonia, p. 80.) When the fluid is in large quantity, moving or talking increases the dyspnea decidedly and there may even be orthopnea, while cyanosis develops or increases. Many cases, however, even with considerable effusion, are not ill enough to desire to be in bed, and may be about without noticeable symptoms or with merely some degree of debility and anemia, loss of flesh, and slight cough and dyspnea. In some cases the onset is much more insidious and the disease is overlooked until effusion is discovered on physical examination of the chest. In still other instances the pleurisy develops as a sequel to pneumonia; although this is oftener true of cases with purulent effusion. The examination of the blood in uncomplicated serofibrinous pleurisy exhibits only a moderate increase in the number of leucocytes or none at all. Any very decided increase points to some complicating pulmonary condition.

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[1] *Loc. cit.*

[2] Jahrb. f. Kinderh., 1898, XLVII, 333.

Physical Signs.—The results of physical examination are very characteristic in typical cases of effusion of considerable size. Inspection shows evident distention of the affected side, with broadening of the costosternal angle, fullness of the interspaces, and decidedly diminished expansion. Palpation reveals diminished or absent vocal fremitus, although this is of less value in early life, distinct fullness and resistance in the intercostal spaces, and displacement of the apex-beat of the heart to the left when a large effusion is on the right side of the chest, and more decidedly to the right in left-sided effusion, the impulse then being discoverable below the ensiform cartilage. The percussion-signs vary with the degree of effusion. Where this is very large there is entire flatness over the whole side, with very characteristic increased resistance to the finger. In less marked effusion the lower portion of the chest gives a flat note, while above this is only dullness, or a Skodaic tympanitic note; and toward the apex in front there may be a hyper-resonant tone. Posteriorly on the healthy side there is usually found a narrow triangular area of dullness (Grocco's sign) with its base to the spine and its upper angle about on a level with the upper layer of the dullness upon the diseased side. If the effusion is upon the right side of the thorax the lower border of the hepatic dullness may be depressed; if on the left the normal cardiac dullness is displaced to the right of the sternum, and in Traube's semilunar space dullness replaces the tympanitic gastric resonance. A shifting of the line of percussion-dullness can often be elicited by change of position. Auscultation exhibits physical signs of considerable variety, and leads to many errors in diagnosis. Theoretically there should be absent or feeble respiration and diminished vocal resonance or one with an egophonic character, and these are, in fact, the signs present in many cases; but in many others in children there is decided bronchial respiration and bronchophony heard over the fluid, of an intensity which makes one readily suspect the presence of pneumonia. In still others, even with considerable effusion, the breath-sounds and voice-sounds may show very little alteration. It is not uncommon, too, for mucous râles to be transmitted through a layer of fluid of sufficient thickness to produce dullness on percussion. Friction-sounds may be heard in regions above the position of the effusion. Always in the case of free fluid, with the patient in the sitting position, the greatest amount of it will be found at the base posteriorly, a thinner layer spreading upward over the lung behind and in the axilla, and to the lower part of the thorax in front. As the secretion increases in amount the evidences of it in the thorax become more evident in all directions, the most marked signs still always remaining in the lower portion of the chest. In cases where s encapsulate the fluid in positions differing from the ordinary physical signs are correspondingly changed.

Complications.—Except for the presence of pneumonia these are few. Sometimes the peritoneum or the pericardium share in the process, especially if of a tuberculous or of a rheumatic nature.

Course and Prognosis.—In the great majority of cases the fluid does not become excessive in amount and is gradually absorbed. Usually evidences of its presence are discovered in 2 or 3 days or within a week from the onset. In other instances an insidious onset does not permit of any determination of the duration. Absorption may take place in 2 or 3 weeks, but often requires a much longer time, especially when the disease is of a tuberculous nature. Barthez and Sanné[1] in analyzing 278 cases found the duration 3 weeks or less in 47 per cent., the remainder ranging from 1 to 7 months. In those in which absorption is much delayed there is liable to develop more or less debility, anemia, and irregular fever. In these prolonged cases treatment seems to have little influence upon the course, and aspiration may be succeeded by a slow reaccumulation of the fluid. Sometimes, however, the aspiration of a portion of the fluid will be followed by rapid absorption of the remainder; and even without the operation recovery eventually takes place in most instances. In all cases recovery is prone to leave adhesions persisting to some extent, although these are seldom a source of trouble later, unless the pleurisy has been unusually severe and prolonged. In such instances evidence of diminished expansion of the lung may be present, with slight impairment of percussion resonance, and this condition may last for many months unless relieved by treatment. There is also a danger of the effusion early becoming purulent. This is true particularly of the cases secondary to pneumonia in infancy or early childhood. It is only very exceptionally in children that the amount of serous effusion is great enough to endanger life by pressure upon neighboring organs, especially the heart and great vessels. In these there is increasing dyspnea and cyanosis, attacks of syncope, and even sudden death.

Diagnosis.—The recognization of the presence of fluid in the thoracic cavity rests principally upon the dullness or flatness on percussion which may shift with change in the position of the patient; diminution of movement of the chest-wall; fullness of the interspaces; and, when discoverable, absence of vocal fremitus over the dull area and its presence elsewhere. Displacement of the heart is a valuable sign when present. Dullness in the paravertebral triangle aids in distinguishing from pneumonia. Among suggestive auscultatory signs are distant bronchial respiration and voice-sounds. The evidences of serofibrinous pleurisy are, however, so equivocal that the distinguishing of effusion from pneumonic consolidation and certain other conditions is far from easy. This applies as well to purulent effusion, in which the physical signs are the same. The differentiation of effusion of either kind from pneumonia or other diseases with similarly confusing physical signs will be discussed under the diagnosis of purulent pleurisy (p. 111). Here only may be said, as regards symptoms in general, that the onset of serofibrinous pleurisy is less severe than that of pneumonia, and the evidence of illness in all respects less marked except the pain in the chest, which is usually much more intense. Later in the subacute stages the presence of dyspnea with little or no fever points to the existence of serous fluid. Always the exploratory needle should be used to confirm the diagnosis; for not only is it important to distinguish between pneumonia and pleural effusion, but still more so to know whether or not the effusion is serous or purulent, and none of the physical signs are capable of determining this.

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[1] *Loc. cit.*, 849.

Treatment.—In the early stages this is simple and is similar to that required for dry pleurisy, including the relief of pain by mustard plasters, dry cupping, ice-bags, phenacetin, or opiates if necessary; rest in bed; suitable light diet, chiefly liquid; and remedies to relieve fever if required. After the acute symptoms are over and the amount of fluid has become decided, measures must be taken to promote absorption. Keeping the skin of the thorax irritated with iodine is a favorite method for older children. Internally the salicylates have been recommended, and the administration of diuretics and laxatives is certainly useful. The curtailing greatly the amount of fluid ingested, at the same time that the patient is purged daily, is an efficient treatment in adults, but is almost too distressing for employment in children. In all cases of considerable effusion exercise must be avoided, since fatal syncope sometimes follows abrupt movement. All such cases should be kept in bed, and even sudden sitting up forbidden. In effusion of less amount, in which recovery is delayed, the patient may with care be placed in a chair and taken into the open air as much as possible. Indeed, the fluid is often so slow in disappearing that all possible measures must be adopted to improve the general condition of health. The food is now no longer liquid but of the most strengthening kind. The iodides internally are sometimes of value in long-continued cases, and tonic remedies are indicated, iron often being needed for the resultant anemia.

It is only exceptionally that thoracentesis is required in cases of serofibrinous effusion, as where a rapid increase of the exudate threatens life, or where the amount of effusion remains unchanged in spite of medical treatment. Sometimes in such cases removal of a small amount of the fluid appears so to alter the conditions that the remainder is soon absorbed. In some instances several such aspirations may be necessary. Incision and drainage have been occasionally performed, but are certainly less often required than is aspiration. For this purpose a Potain aspirator is satisfactory, with a small trocar and canula. The child should be in a sitting position; the skin, instruments and hands of the operator sterilized in the usual manner, and the needle inserted by preference in the 7th interspace in the posterior axillary line, or elsewhere if the fluid is unusually situated. The fluid should be withdrawn slowly, and if the chest is very full, no attempt should be made to remove the entire amount, or faintness may develop. In cases with less fluid the aspiration may continue until the flow ceases or troublesome coughing develops, although this completeness of removal is not necessary. The needle is then withdrawn and the puncture closed by antiseptic gauze or by cotton and collodion. After recovery from long-continued serous effusion treatment is required for the persistent, firm adhesions and the diminished expansion of the lung which results. Here pulmonary exercises are useful, including such as swinging from a horizontal bar, vigorous horse-back riding, and the like. In one instance I found benefit come from having a country child pump water for the household.

3. PURULENT PLEURISY

(Empyema)

Etiology.—The symptoms and lesions of this form of pleurisy depend to a certain extent upon the nature of the germ present. Cases immediately following pneumonia are generally due to the action of the pneumococcus alone or sometimes associated with other germs. These, as already stated (p. 100) form the decided majority of cases of empyema in early life. Pneumococcal pleurisy may exceptionally be encountered, however, as a primary disease without previous pneumonia. Cases following the infectious diseases, such as scarlet fever, measles, erysipelas, and diphtheria, depend oftener upon the streptococcus, or a combination of this with the pneumococcus or less often with some other germ. Empyema occurring as one of the manifestations of sepsis is most frequently due to the streptococcus or less frequently the staphylococcus. The tubercle-bacillus is an uncommon cause of purulent pleurisy in early life.

Pathological Anatomy.—The lesions differ from those of serofibrinous pleurisy in that large numbers of pus cells are present in the fluid. When the staphylococcus or the streptococcus is the causative agent the fluid may be purulent from the beginning, contains less fibrin, is thinner and of a greenish-grey color, and separates into layers on standing. In the cases due to the pneumococcus the pus is thick, tenacious, green, does not separate on standing, and contains a large amount of fibrin. Sometimes it is at first apparently serofibrinous, and more or less rapidly becomes purulent through an early increase in the number of pus cells. These distinctions between the two varieties of pus are not, however, absolute. Not uncommonly the purulent fluid is more or less tinged with blood (*hemorrhagic empyema*).

At first the effusion is encapsulated by adhesions, and may remain so; but if the amount is large these give way and the thorax fills on the affected side, which is oftenest the left. If encapsulated, any part of the chest may be affected; if free, the fluid is in largest amount at the base posteriorly as in cases of sero-fibrinous effusion. *Bilateral empyema* is exceptional. Hellin[1] collected 114 cases from medical literature. Of 52 in which the age was definitely stated, 40 were in subjects from 8 months to 12 years. Fabricant[2] collected 117 cases of all ages of double empyema, besides 1 of his own, and found it commonest in childhood, and in about ¾ of the cases following pneumonia. Statistics vary as regards its relative frequency. Blaker[3] observed it 9 times in 81 cases of empyema, while Koplik[4] saw only 3 cases in 170. It is observed most frequently in infancy.

In cases of empyema where the fluid is free a layer extends upward over the back of the lung, and often still higher in the axilla, and to a less extent in front, gradually diminishing in thickness the higher its position. When the amount is large the lung is compressed in the direction of its root and becomes almost airless (Fig. 300). The other organs are affected as in serous effusion. The amount of fluid present in purulent pleurisy varies greatly. Sometimes small encapsulated effusions contain only 2 to 4 ounces (59 to 118). In other instances the quantity may equal several pints. In cases in which recovery takes place under proper treatment adhesions will remain, but give no trouble except where the duration has been long; in which event contraction of the chest may occur. This is much oftener the case than with serous effusion.

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[1] Berl. klin. Woch., 1905, XLII, 1415.
[2] Deut. Zeitschr. f. klin. Chir., 1910–11, CVIII, 584.
[3] Brit. Med. Jour., 1903, I, 1200.
[4] Diseases of Infancy and Childhood, 1910, 652.

Symptoms.—In the large majority of cases, in which the disease follows upon a pneumonia (*metapneumonic empyema*), the temperature of the primary affection often falls to normal and may remain so for several days and then begins to rise again, exhibiting an irregular course (Fig. 301). Sometimes, however, no distinct intermission occurs between the pneumonia and the empyema, but the fever continues, showing only the change in character by its irregularity. In some of these the development of the empyema is coincident with that of the pneumonia (*parapneumonic empyema*). This is much less common than the metapneumonic form. Where there has been no preceding pneumonia, as for instance after the infectious diseases, or in the cases developing without scoverable cause whatever, the onset of empyema is marked by

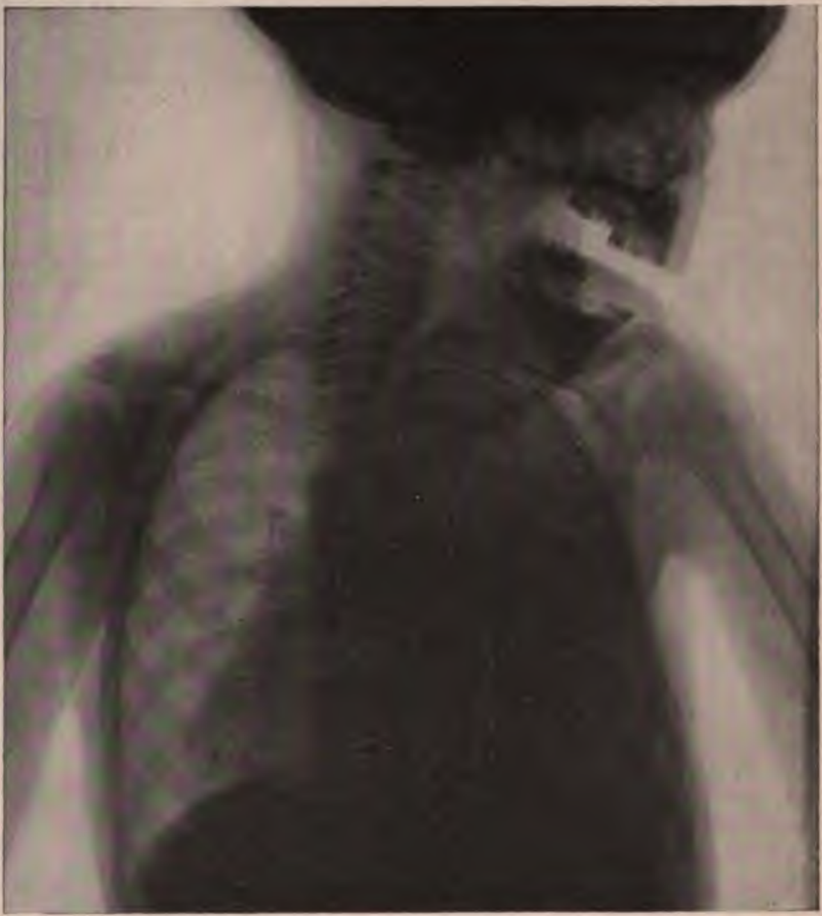

FIG. 300.—RADIOGRAPH OF PLEURAL EFFUSION.

Jessie G., aged 6 years. Admitted to the Children's Medical Ward of the Hospital of the University of Pennsylvania, June 2. Had suffered from cough for 2 weeks, pain in right side. *Examination.*—Moderate prostration, decided dyspnea, right side of chest shows obliterated interspaces and diminished expansion and breath-sounds, percussion flat below 4th rib, leucocytes 21,000, exploratory puncture gave yellow pus. Viewed from behind.

the development of irregular fever, increased rapidity of the pulse and respiration, dyspnea, cough, debility, anemia, loss of appetite, emaciation, and a decided leucocytosis, principally polymorphonuclear, reaching 20,000 to 50,000 or over. Diarrhea may occur, especially in the cases dependent upon the streptococcus. As a rule the fever is higher than in cases of serous effusion.

After a varying time the acuteness of the symptoms is prone to subside, fever becomes less or may be absent altogether, or there may be only occasional elevations and the pulse may be less rapid although continuing feeble and accelerated. There is, however, little if any improvement in the general symptoms, and the dyspnea, anemia, debility, sweating, and signs of general cachexia persist. In long-continued cases there may be enlargement of the spleen and liver, albuminuria, enlargement of the terminal phalanges, and general edema. The unusual condition denominated *pulsating empyema* has occasionally been observed in children. In Wilson's[1] series of 68 cases, 7 occurred in the first 10 years of life. The effusion in purulent cases generally develops rapidly, and the physical signs then do not differ from those characteristic of serous effusion as already described. In many instances, however, it is localized and then very difficult of discovery. Thus it may occasionally be of small size and near the apex; between two of the lobes of the lung; between the lung and the diaphragm, or in the mediastinum.

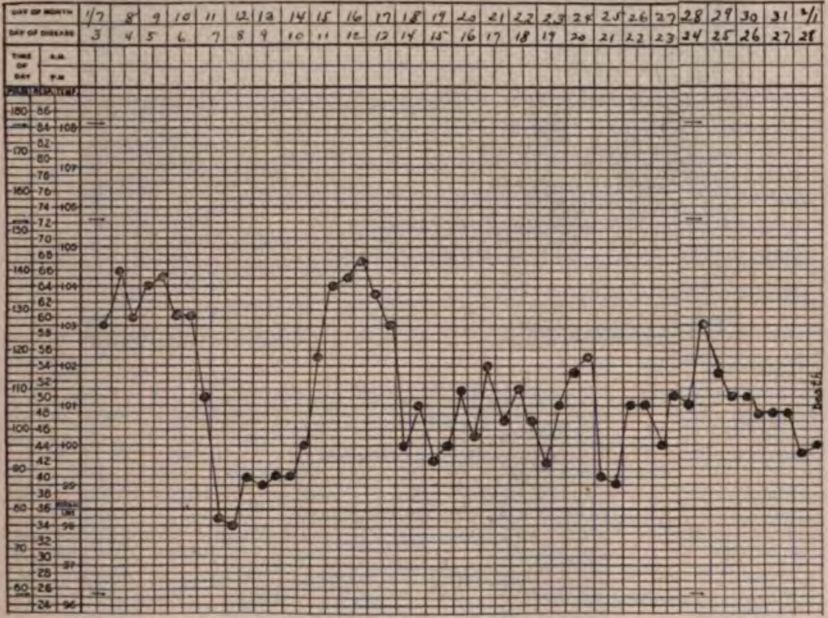

FIG. 301.—EMPYEMA, FOLLOWING PNEUMONIA.

Fannie B., 2 years old. Admitted to the Children's Ward of the University Hospital, Jan. 7. Croupous pneumonia, with crisis on the 7th day. Rise of temperature began on the 10th day. Aspiration showed pus on the 11th day, and operation done on the following day. Gradual failure of strength, and death on the 28th day.

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[1] Transac. Assoc. Amer. Phys., 1893, VIII, 195.

Complications.—Purulent pericarditis may occur in the cases of left-sided pleural effusion; or an abscess of the lung form; or meningitis, arthritis, peritonitis, or other manifestations of septic involvement extend from the pleura as a centre. In 29 fatal cases of empyema Wightman[1] recorded pericarditis in 12 instances, 10 of them being purulent. Tuberculosis of the lungs may develop in older children. A pneumothorax sometimes forms if the pus communicates with a bronchus. Bronchopneumonia may occur and be a serious complication.

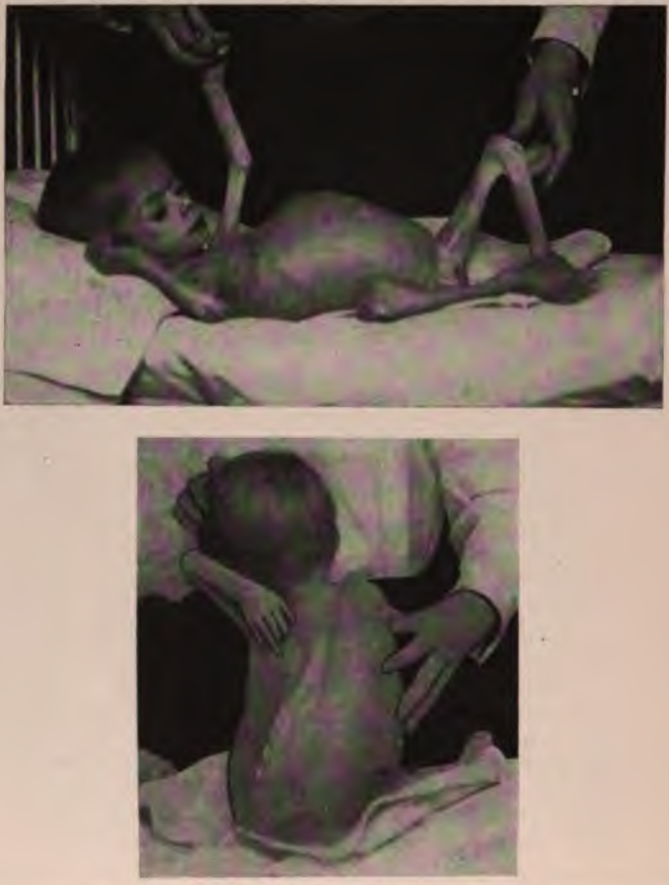

FIG. 302.—CHRONIC EMPYEMA.

Child of 3½ years, weight 8 pounds, 14 ounces (4026), with long-standing empyema of the left side. (*Hand, Arch. of Pediat.*, 1910, *XXVII, Jan.*)

Prognosis and Course.—In the great majority of cases not relieved by treatment the course is progressively toward a fatal ending. The debility, anemia and prostration continue to increase and fever persists, unless the pus finds a way out by perforating a bronchus, or oftener by breaking through the chest-wall (*empyema necessitatis*). When about to perforate externally a fluctuating, red swelling appears between the ribs, usually in front near the nipple. In other instances the pus perforates iaphragm and enters the peritoneum.

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[1] Lancet, 1895, II, 1357.

The duration of the disease before death occurs is very indefinite. It is possible in infancy for a chronic empyema to exist unsuspected for many months, the case having the appearance of marasmus, or being attributed to tuberculosis. In a case in the Children's Hospital of Philadelphia reported by Hand[1] (Fig. 302) the disease had lasted 18 months, and the child of 3½ years weighed but 8 pounds 14 ounces (4026). It happens only very occasionally that a small purulent exudate is absorbed and recovery follows without treatment. Under surgical intervention, the only treatment applicable, the prognosis is influenced by various circumstances. In infancy the mortality is high even when operation has been done and other conditions are favorable. In the 145 cases in children reported by Nathan[2] 46 were under 1 year, and of these 45 died, a mortality of 97 per cent. Of 30 in the 2d year, 18, or 60 per cent., died. In 154 cases reported by Holt[3] the mortality for the 1st year was 73 per cent. and for the 2d year 58 per cent. After the 3d year the decided majority of cases will do well if the disease is not too far advanced. The general mortality of operative hospital cases, many of them, of course, already well advanced and unfavorable, varies considerably, but would appear to average from 10 to 16 or more per cent. (Netter).[4] In the collected statistics of Wightman[5] in 656 cases there was a mortality of 16 per cent.; and in 285 cases reported by Dowd,[6] all but 47 in children and chiefly between 2 and 14 years of age, the mortality was 25.6 per cent. The nature of the germ present is of great prognostic importance. That empyema gives on the whole a much more favorable prognosis in early life than later is due to the fact that the pneumococcus is much oftener the cause at this age. The prognosis of streptococcic empyema at any time of life is unfavorable. Tuberculosis, the cause of many fatal cases of purulent pleurisy in adults, is exceptionally the active agent in early life. The prognosis of bilateral empyema is unfavorable. The mortality in Hellin's[7] cases was 30.1 per cent. Of course, the previous state of the health and the condition of the child at the time of operation for empyema possess important prognostic significance. The presence of complications influences the mortality unfavorably. Pericarditis is especially to be mentioned here.

The final recovery from empyema in children, if operated upon early, is usually remarkably complete, even when the effusion has been large. The lung expands fully, and little if any retraction of the chest or curvature of the spine remain after the lapse of some months or years. The longer operation is delayed the greater is the danger of unyielding adhesions persisting, with failure of the lung to expand and with consequent contraction of the thoracic walls. The duration of the disease after operation has been done varies greatly, depending upon the general health of the patient and the amount of the effusion. About 6 to 8 weeks might be allowed for the favorable cases with recovery, but the time may extend much beyond this.

Diagnosis.—First is to be determined that an effusion exists. The recognition of this depends upon the presence of the diagnostic physical signs already discussed under serofibrinous pleurisy (p. 102). They consist chiefly of dullness, which is greater as one goes lower, and becomes flat in the lowest portion of the chest; increased resistance to the percussing finger; diminished movement of the chest-wall; fullness of the interspaces; fremitus absent over the dull region and present above this; displacement of the heart or liver; and absent or distantly bronchial voice- and breath-sounds. Yet in very many cases the diagnosis is one of great difficulty. The fluid may occupy the same region as a preceding pneumonia and be situated in the middle or upper portion of the chest instead of at the base. Changes in fremitus must always be judged guardedly in children. The voice- and breath-sounds may be quite loudly bronchial; the presence of bronchitic râles over an area of dullness by no means excludes a purulent pleurisy; and, in general, auscultatory signs are liable to be very misleading in cases of effusion. This is especially true of infants and young children, in whom the voice-sounds cannot be studied except by the accident of crying, and where it is impossible to command deep respiration. Many times in cases of small purulent effusion the diagnosis is rendered probable only by the history and course of the disease, and repeated exploratory punctures may be needed to confirm it.

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[1] Arch. of Ped., 1910, XXVII, Jan.
[2] Arch. f. Kinderheilk., 1903, XXXVI, 252.
[3] Amer. Med., 1913, XIX, 381.
[4] Traité des mal. de l'enf.; Grancher, 1904, III, 674.
[5] *Loc. cit.*
[6] New York State Journ. of Med., 1914, XIV, 342.
[7] *Loc. cit.*

Pleural effusion either serous or purulent is to be distinguished especially from *pneumonic consolidation*. In acute cases of the latter the voice- and breath-sounds are usually louder; the dullness on percussion more limited; the râles if present more readily transmitted to the ear; and the general condition of the child distinctly worse. A great diagnostic difficulty exists in cases of unresolved pneumonia. Here, as in the subacute stage of pleural effusion, the active symptoms have disappeared, and reliance must be placed upon the physical signs. In pneumonia, even in this stage, the voice- and breath-sounds are usually louder; the consolidation is limited to a portion of the chest and does not increase as time passes; displacement of other organs is absent; there is not the absolute flatness on percussion; no dullness in the paravertebral triangle; no shifting with change of the position of the patient; and there is greater expansion of the side with respiration and less fullness of the interspaces. In *pleuropneumonia* the presence of plastic exudate modifies the signs of ordinary croupous pneumonia and suggests effusion. Yet no displacement of the organs occurs, and less diminution in the expansion of the chest-wall and fullness of the interspaces is seen. Nevertheless in pleuropneumonia, as also in pneumonia with delayed resolution, only exploratory puncture may exclude the presence of empyema. *Hydrothorax* is recognized by the existence of causes sufficient to account for it, together with the presence of cutaneous edema or of ascites; the absence of symptoms of inflammation of the thoracic cavity; and the fact that the fluid usually occupies both sides of the thorax. Fluid obtained by puncture is of low specific gravity, and the few cells present are endothelial rather than leucocytic.

Atelectasis in infants gives rise to dullness on percussion, feeble respiration and often great dyspnea. There is, however, no fullness of the interspaces or other of the usual signs of fluid in large quantity. To exclude the presence of a small amount of fluid exploratory puncture should be made. *Pericardial effusion* if of large amount may closely simulate pleural effusion of the left side, especially as the distended pericardial sac results in an extension of dullness to the right of the sternum as though the heart were displaced. I have, too, seen dullness reach so far to the left that puncture in the axillary region revealed serous fluid, only later found to be derived from the pericardium. Such a condition is, however, rare. *Chronic adhesive pleurisy* produces dullness and feeble respiration suggestive of effusion. There is, however, no distention of the chest-wall, but often retraction. Yet many cases are so doubtful that exploratory puncture should be done.

Most important is the distinction of an *empyema* from a *serofibrinous effusion*, since the prognosis and treatment are so widely different. The physical signs are identical, and only the general symptoms and the history of the case are of service. In infancy the fluid is nearly always purulent; between the ages of 2 and 5 years either form may occur, purulent effusion being rather more common; while after 5 years of age the fluid is much oftener serous. If a case of pneumonia continues to have the temperature elevated after the period when it should normally have fallen; or if after a short afebrile period the fever returns, pleural effusion should always be suspected; and a decided polymorphonuclear leucocytosis of 20,000 or more indicates the probability of the exudate being purulent. A rapid increase of the amount of fluid is also an indication of its purulent nature. With the suggestive history mentioned, empyema should always be suspected even when no physical signs are present, or those of a most equivocal sort. The general symptoms of purulent exudate are also much more severe than those of the serous form. There is more fever at the onset, and later a tendency to a remittent type, although fever may be absent entirely at this time. The anemia, cachexia and progressive loss of health and strength are very suggestive.

Exploratory Puncture.—The diagnosis of the presence of fluid and of its nature is of such great importance that exploratory puncture should be done in every case admitting of the slightest doubt. It is of common experience to encounter cases of empyema terminating fatally merely because of a failure of early diagnosis. The operation is without danger under proper precautions. There is a theoretical danger of sudden death from the procedure in cases with the lymphatic diathesis, but for all practical purposes this can be ignored. The syringe employed for the aspiration should be entirely of metal, or preferably of glass, in order to allow disinfection by boiling. The needle should be of about 1 mm. (0.04 inch) in diameter and stouter and of greater lumen than the ordinary hypodermic needle, since the latter is easily choked by floating portions of fibrin or is too narrow to allow a thick exudate to pass. That designed for lumbar puncture answers well. The needle and syringe should be boiled just before being used, and the operator's hands and the skin of the suspected area of the child well disinfected. For this purpose I generally use soap and water, followed by a 50 per cent. alcohol. Tincture of iodine may be painted on the skin if desired. The apparatus should always be tested before use, to determine that the suction of the piston is good, no air-leaks present, and the needle permeable. The child should be firmly held by the nurse in a sitting position with the arms drawn forward and the healthy side of the chest pressed against her in such a manner that any sudden movement will not break the needle. The site chosen for puncture is that where the physical signs are most suggestive, especially where the dullness is greatest, but avoiding the cardiac or hepatic region. Other things being equal the posterior axillary line in the 7th interspace may be chosen. With the sterilized finger pressed firmly upon the upper border of the rib, the needle is introduced just above this as a guide, thus avoiding both the rib and the blood-vessels running beneath it. A depth of ½ to 1 inch (1.27 to 2.54 cm.) is sufficient. Too short a puncture fails to penetrate the chest-wall and any plastic exudate present; too deep a one may pass through the fluid and enter the lung. The syringe must always be held lightly during the whole process, in order to avoid danger of snapping the needle if the child moves. So, too, the operation should be done rapidly but *never* hurriedly. Traction on the piston may be followed by the appearance of fluid, but sufficient time must be allowed, since a purulent effusion often flows very slowly. After withdrawal the point of puncture is covered with a small piece of adhesive plaster, or a film of cotton may be laid over it and then painted with flexible collodion. Should nothing be found at the first puncture in cases where the presence of pus seems probable, a second or a third should be immediately made in another spot, and this, if unsuccessful, repeated in a few days. The pain to the child is little greater than that of an ordinary hypodermic injection, and a local anesthetic is hardly required.

Treatment.—Apart from the general tonic remedies, sustaining diet, stimulants, and the like, needed for any debilitated state, the treatment of purulent pleurisy is entirely surgical and consists in the removal of the pus as promptly as possible. In a few cases aspiration of the pus is sufficient, but is on the whole so unsatisfactory that it is not to be recommended. When the collection of fluid is quite small and there are few characteristic symptoms present, a single aspiration may be tried, but if the symptoms persist and the fluid returns, more radical measures are necessary, and it is questionable whether it is not always better to employ these at the outset. The procedures recommended are surgical in nature and to a certain extent beyond the province of this discussion. Merely may be stated here that of the various methods employed the principal are simple incision followed by insertion of a drainage tube; incision followed by resection of a rib and then drainage with the tube; and incision with subsequent siphonage of the pus through a long tube passing into a vessel of water, thus preventing the ingress of air. Simple incision and drainage are sufficient in many cases in infancy. In children past this period resection of a rib is advisable to prevent the subsequent pinching of the drainage tube by the ribs on each side and the interference with the discharge of pus. Many surgeons recommend resection at any age. Siphonage is useful at any period of childhood but is especially serviceable in infancy. In the case of very ill children, or indeed for any infantile patient, only local anesthesia should be employed, since general anesthesia is not without danger of sudden death on the operating-table, or of being later productive of bronchopneumonia. Older children in better condition usually require a general anesthetic.

After the operation there is commonly prompt relief of the symptoms. If the fever continues or returns after an interval, it is usually an indication that drainage is not sufficient, or that some complication is present. To facilitate the expansion of the lung during convalescence in cases where decided adhesions have formed, the children may be taught to employ forced expiration, as by blowing soap-bubbles or by driving by the breath colored fluid from one Wolf-bottle fitted with a glass-tube into another connected with it by rubber tubing. In long-continued chronic cases in older children, where the adhesions are too great to be remedied in this way, Estlander's operation of removing a considerable portion of several ribs may be necessary, to permit of the closure of the empyemic cavity. The administration of autogenous vaccines has been employed, and is deserving of further trial.

HYDROTHORAX

This is only a symptom of other affections. It consists in the accumulation of liquid which is not of an inflammatory character; a transudate in contradistinction to an exudate. It is of somewhat lower specific gravity, than the fluid of sero-fibrinous pleurisy, more watery, contains much less albumin, no bacteria or leucocytes, and does not form a fibrin-clot on standing. Hydrothorax is nearly always bilateral, although commonly greater on one side than the other; and is usually accompanied by the escape of fluid in other regions, as the peritoneum, the pericardial cavity, and into the subcutaneous tissues. Its most frequent cause is disease of the heart or kidneys, although it occasionally occurs in combination with other effusions in cases where no cause whatever can bo discovered; and in that event may finally disappear completely and permanently. Rarely it may be produced by pressure upon the veins. Fever, cough, pain, and friction-fremitus are absent; and any symptoms present are produced by the primary disease, with the exception of those the direct result of the mechanical interference by the fluid. Dyspnea and cyanosis are prominent among these, although they may be caused chiefly by the primary affection. The physical signs are those of fluid found in the pleural cavity, with ready shifting of the level of dullness by change of position.

The treatment is that of the primary disorder. In addition aspiration may be necessary if the symptoms produced by pressure are threatening.

CHYLOTHORAX

This is not a common condition at any time of life, and in children is rare. Its occurrence at this period has been studied especially by Sherman,[1] who analyzed 11 cases, including 1 of his own. It depends upon the escape of more or less chyle from the thoracic duct or its branches into the thoracic cavity. This is probably oftenest produced by trauma with subsequent rupture, but apparently may sometimes be a transudation, brought about by pressure of enlarged bronchial glands upon the duct, or in other ways. In certain cases a milky fluid is obtained which is due to a fatty degeneration of formed elements, and not to a true effusion of chyle. In these cases there are numerous leucocytes present. Such a fluid is *chyliform* in contradistinction to truly *chylous*, and is not dependent upon the thoracic duct. Such effusions do not clear when shaken with alkali and ether.

The symptoms and physical signs are those incident to the presence of fluid in the thoracic cavity. The characteristic of chylothorax is the discovery by puncture of a chylous effusion containing fat. There may also be present sugar, albumin, cholesterine, lecithin and a few lymphocytes. The prognosis is grave. Of Sherman's series of 11 cases, 4 died, and the result was not known in 2. Treatment is in part expectant and supporting. Aspiration may be needed to relieve the pressure, and may be attended by permanent relief.

PNEUMOTHORAX

Etiology and Pathological Anatomy.—This is a very uncommon affection in early life. West[2] in 98 cases at all ages saw only 3 at less than 15 years of age; Bovaird[3] could collect but 18 cases in children in the 10 years up to the year 1903, including 5 reported by himself. Tuberculosis, so commonly the cause of the disease in adults, is rarely the active agent in childhood. In only 3 of the 18 cases in Bovaird's series was tuberculosis observed. Oftener it is seen after pneumonia (Fig. 303), whooping cough, measles, diphtheria, or emphysema, in which coughing occasions a rupture of a vesicle into the pleura; or occurs in connection with empyema, or with abscess or gangrene of the lung which has broken through the pleura, in this event producing a pyopneumothorax. Trauma of the chest with fracture of a rib may be a cause. Naturally the incision of the thoracic wall in cases of empyema admits air to the pleural cavity, but this operative pneumothorax is generally limited to a certain extent by adhesions, is but temporary in favorable cases, and is not considered here.

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[1] Arch. of Ped., 1907, XXIV, 646.
[2] Lancet, 1884, I, 791.
[3] Arch. of Ped., 1903, XX, 817

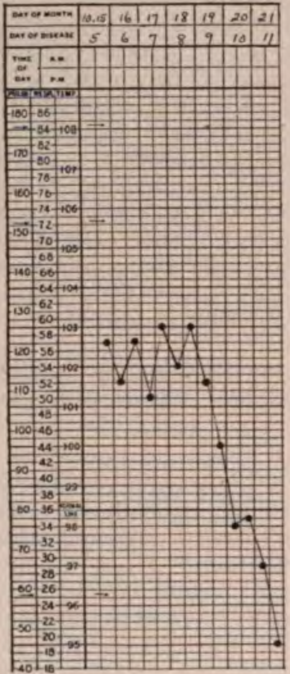

FIG. 303.—PNEUMOTHORAX FOLLOWING CROUPOUS PNEUMONIA.

Annie G., aged 3½ years. Crisis of pneumonia began on the 10th day, at which time typical physical signs of pneumothorax were discovered. Child failed and died on 12th day. Autopsy showed croupous pneumonia, a fibrinous patch on the pleura, and a small perforation here passing into a small bronchus and producing a pneumothorax.

The pathological lesions are those observed in adult life. The lung collapses and is drawn backward toward its root, unless adhesions already present prevent this, and limit the lesion to a small area. The entrance of germs with the air is liable soon to transform a simple pneumothorax into one in which serous or oftener purulent effusion is also present.

Symptoms.—The symptoms and physical signs may not differ materially from those seen in later life, except that the latter are usually more difficult of recognition, and the former are often more obscure. If the pneumothorax is limited in extent, displacement of neighboring organs does not occur, and the development of the disease may in fact be insidious. In other cases, however, the onset, symptoms and physical signs are those typical of later life and there is the sudden occurrence of dyspnea, prostration, cyanosis, widespread tympany, faint amphoric breathing, and the bell-percussion.

Diagnosis.—Pneumothorax is to be distinguished especially from emphysema, resembling it in the tympanitic resonance and feeble respiration, but differing in the amphoric breathing. Careful examination will readily distinguish it. The fact that emphysema occurs on both sides of the chest is of importance. If the air in pneumothorax is under high compression a dull note on percussion may be produced and pleural effusion may be suspected. A high situation of a dilated stomach may on first examination suggest pneumothorax, but can be recognized readily with care.

The **prognosis** of pneumothorax depends upon the cause. When this is not one which occasions repeated entrance of air into the chest, or which is productive of pus, simple pneumothorax will usually recover. The tendency, however, is for pyopneumothorax to develop.

Treatment other than symptomatic is only needed in those cases in which the pressure of air is sufficient to threaten life, or where a pyopneumothorax exists. In the former aspiration is necessary. In the latter the surgical treatment for empyema is required.

MALFORMATIONS, PARASITES, AND MORBID GROWTHS OF THE LUNGS AND PLEURA

Hernia of a portion of the lung through a defect in the thoracic wall is a rare congenital anomaly. It presents an elastic tumor covered by skin and pleura, gives a resonant percussion note and is reducible by pressure. **Hydatid cysts** of the lung are very unusually seen in later childhood in geographical regions where this parasite is encountered. Infection of this part of the body stands next to that of the liver in frequency. The symptoms are indefinite and the disease usually not recognized unless there is associated involvement of the liver, or unless aspiration and microscopical examination are made, or the evidences of the disease are discovered in the expectoration. An eosinophilia may be present. The symptoms consist of cough, dyspnea, dullness on percussion, and occasionally a prominence in the affected region. **Actinomycosis** is very exceptionally observed in this country. It exhibits symptoms suggesting a neoplasm or empyema. Only microscopic examination of any pus produced and discharged can establish a diagnosis. **Malignant neoplasms**, especially sarcoma, are sometimes seen as a secondary development in the lung or pleura, or very rarely primarily there.

SECTION VI

DISEASES OF THE CIRCULATORY SYSTEM

CHAPTER I

INTRODUCTORY

The characteristics of the anatomy and physiology of the heart and blood-vessels in early life, including the arrangement of blood-vessels in the fetus, have already been discussed. A brief description of the circulation in fetal life is required before the subject of congenital anomalies is reviewed. Further consideration must also be given to some of the diagnostic indications of symptoms especially connected with circulatory disease.

INDICATIONS DERIVED FROM THE PHYSICAL EXAMINATION OF THE HEART IN EARLY LIFE

In addition to the data regarding the size and position of the heart and the percussive and auscultatory phenomena already reviewed to some extent (Vol. I, p. 54), some short further consideration of the indications of the physical signs will be of service.

The fact that the percussion dullness of the heart is relatively greater in early life than in adults (Vol. I, p. 55) is a matter not to be forgotten in making a diagnosis of pathological conditions. Equally important is the knowledge of the normal position of the apex-beat at this period. Thus in infancy an apex-beat found in the 5th interspace is abnormally low by one interspace, and it is only after the 7th year that it is discovered nearly always in this position. Normally, too, the apex-beat is farther to the left than in adult life, and a situation beyond the mammillary line is not an evidence of enlargement (Vol. I, p. 55). It is particularly in early life, owing to the yielding character of the thoracic framework at this time, that cardiac enlargement is so often productive of very decided precordial bulging. Irregularity of the cardiac rhythm is a normal feature of infancy and to some extent of early childhood, especially during sleep. As a pathological manifestation it may occur during convalescence from infectious disorders; in toxic conditions from intestinal and other sources; in intracranial affections, and in maladies of the heart itself. Reduplication of the heart-sounds observed at this period is frequent and is likewise not necessarily an indication of disease. Slowness of the heart's rhythm is often pathological. It may be due to various toxic agents, as seen, for instance, in jaundice and diphtheria; and may occur also in endocarditis, diseases of the brain, and heart-block; but is also not uncommon in convalescence from fevers and often possesses no special significance. (See Bradycardia, p. 157.) Increased rapidity of the heart's action, on the [illegible] hand, is of such frequent occurrence that by itself it possesses little [illegible]al meaning. Among auscultatory phenomena, of most importance is to remember the normal accentuation of the pulmonary second sound, which is louder in early life than is the aortic. This is especially true during infancy and early childhood. The first sound of the heart is always louder than the second during the first 4 or 5 years of life and in the 1st year is very valve-like, the rhythm suggesting the ticking of a watch. This, so serious a sign of great cardiac weakness in adults, is therefore of no pathological importance at this earlier period. The diffusion of murmurs, as well as of the normal sounds, is often very extensive. This is dependent upon the small size of the chest and the comparatively large mass of the heart. Naturally, therefore, fewer inferences can be drawn from this diffusion than is the case in adult cardiac disease. The accidental cardiac murmurs so common in adult life are claimed to be less often heard in children and rarely in infancy. (See Accidental Murmurs, p. 158.) In my own experience they occur on the whole not at all infrequently, especially as the result of anemia, or they may be cardio-pulmonary in nature. The anemic murmurs are often loud, systolic, and very suggestive of a valvular lesion. In addition to these murmurs may be others audible in the arteries and veins especially of the neck. Those in the arteries are usually merely transmitted from a heart affected by valvular disease. Care must be taken not to exert too great a pressure with the stethoscope or a murmur by compression may be produced. This is, in fact, true even of the heart itself, where firm pressure in rachitic subjects may readily give rise to a systolic murmur. Venous murmurs are common in cases of anemia at all ages. They are oftenest heard in the neck, but sometimes over the front of the thorax also, and may be developed over the manubrium of the sternum when the head is thrown back. It is to be noted, however, that a venous murmur may occur in individuals not anemic. Sawyer[1] and Landis and Kaufman[2] believe it to be present in the majority of children. (See p. 161.) The employment of radiography is of service in the examination of the heart, for the purpose of confirming or amplifying the results of the examination by percussion. The polygraph and electro-cardiograph have been purposely omitted from consideration, partly because they have not as yet rendered the service in pediatrics which they have done in adult medicine; largely because they require much training for their satisfactory employment and for the interpretation of the results obtained.

FETAL CIRCULATION AND THE CHANGES AT BIRTH

The fetal circulation (Fig. 304) has an intimate relationship to the production of congenital diseases of the heart. During intrauterine life the smaller, pulmonary circulation is not in action, since aeration through the lungs is not possible or necessary, but takes place through the placenta. The blood enters the right auricle from two directions: (1) from the superior vena cava which carries blood of a wholly venous character; and (2) from the inferior vena cava in which the blood is of a mixed quality, being composed, namely, of the venous blood from the body of the child and the oxygenated blood brought from the placenta by the umbilical vein. The greater part of the blood from the umbilical vein circulates through the liver before entering the inferior cava; the smaller quantity enters the cava directly from the umbilical vein. After entering the right auricle the partly oxygenated blood from the inferior cava passes to a large extent, according to the generally accepted view, through the foramen ovale into the left auricle and thence to the left ventricle and into the aorta. The wholly venous blood from the superior cava, on the other hand, passes from the right auricle into the right ventricle and thence into the pulmonary artery whence it goes partly, although in but small amount, to the lungs, but chiefly through the ductus arteriosus into the aorta. Since the ductus arteriosus joins the aorta beyond the point of origin of the innominate and the left carotid and subclavian arteries, it follows that these vessels are supplied with blood oxygenated to a greater degree than it is after the entrance of the purely venous blood from the ductus arteriosus has taken place. The blood of the body returns from the general circulation to the placenta by way of the umbilical arteries.

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[1] Brit. Jour. Dis. Child., 1910, VII, 310.
[2] Arch. of Ped., 1912, XXIX, 88.

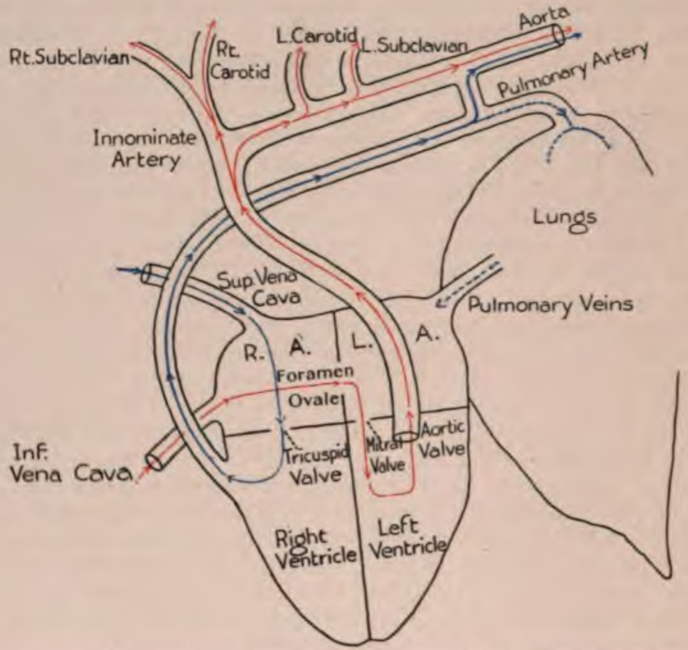

FIG. 304.—DIAGRAM ILLUSTRATING THE COURSE OF THE BLOOD-CURRENTS IN THE FETAL CIRCULATION.

Red, partly arterial blood; *blue*, venous blood.

With the cessation at birth of circulation in the umbilical arteries and vein and the simultaneous beginning of the respiratory function, the pressure in the right heart is much diminished, and, as a result, blood ceases to pass through the foramen ovale and the ductus arteriosus, and both of these close; the latter in the course of 1 to 2 weeks, the former often not until after some months have elapsed, or during the 2d year (Hinze),[1] while not infrequently a small opening remains during life.

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[1] Berlin Dissert., 1893.

CHAPTER II

CONGENITAL CARDIAC DISEASE

This is a condition encountered repeatedly in every large hospital or private practice, although much less frequently than is acquired disease of the heart. Norris[1] in collected statistics of 1272 cases of cardiac disease, reported 97 congenital, a percentage of 7.6. It is observed much oftenest in early life, since the majority of patients die before reaching maturity. In 2792 sick children under 10 years of age, Still[2] observed the symptoms of congenital heart-disease in but 16 instances (0.57 per cent.). These figures would not include the frequent instances of small patulous foramen ovale discovered only at autopsy.

Etiology.—This has been much discussed, and is even yet not clearly understood. The effect of constitutional disease of the parents is questionable, although to syphilis has often been assigned a predisposing influence, which it probably has to some degree. The finding by Watson[3] of a positive Wassermann reaction in every one of 16 cases examined is suggestive, although not conclusive; and there is much evidence against a close causative relationship. Hochsinger,[4] for instance, observed only 7 cases of congenital heart-disease in 500 children with hereditary syphilis. That any cause influencing harmfully the health of the parent is in a way a predisposing factor is indicated by the very frequent coincident occurrence of other defects in development, such as atresia of the anus, harelip, idiocy, polydactylia, and many others. A certain family tendency has been observed. Thus de la Camp[5] reported congenital disease of the heart in 6 members of one family. Statistics collected by Vierordt[6] show a preponderance of cases among males.

As regards the nature of the active cause opinions have been at variance, and more than one cause certainly exists. Many cases are due entirely to an *interference with the normal process of development*, although the reason why this took place is not clear. Here is without question to be included defect of the ventricular septum. Certainly other forms may be due to a *fetal endocarditis;* yet how often this is true is a matter of dispute. In this category belong the cases of stenosis of the valvular orifices, at least in part; others probably being produced by arrested development. Still other instances of congenital heart-disease depend upon a *failure of closure after birth of normal prenatal openings*, this being the result of some other diseased condition of the heart present. In this category are certainly to be placed patulous ductus arteriosus and patulous foramen ovale. It is to be remembered that an endocarditis often may develop as a secondary lesion in extrauterine life, or in intrauterine life be secondary to an arrest of development.

Pathological Anatomy.—Looked at from a standpoint other than that of cause, the usual lesions of congenital cardiac disease may be divided into: (1) Anomalies of the septa between the chambers of the heart; (2) Anomalies of the vessels or their valves; (3) Anomalies in the size or position of the heart. In tabular arrangement the subdivisions of these chief classes are as follows:

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[1] Arch. of Ped., 1906, XXIII, July.
[2] Common Disorders and Diseases of Childhood, 1909, 472.
[3] Glasgow Med. Jour., 1913, LXXX, 303.
[4] Pfaundler und Schlossmann Handb. d. Kinderheilk., 1906, II, 424.
[5] Allgem. med. Central-Zeit., 1902, LXXI, 665.
[6] Nothnagel, Spec. Path. und Therap., 1901, XV, II, 47.

TABLE 84.—CLASSIFICATION OF FORMS OF CONGENITAL CARDIAC DISEASE

1. ANOMALIES OF THE SEPTA	Patulous foramen ovale Perforate septum ventriculorum	
2. ANOMALIES OF THE VALVES AND VESSELS	(a) Valves	Pulmonary stenosis Pulmonary insufficiency Aortic stenosis Aortic insufficiency Mitral stenosis Mitral insufficiency
	(b) Vessels	Patulous ductus arteriosus Stenosis of pulmonary artery Dilatation of pulmonary artery Narrowing of aorta Dilatation of aorta Hypoplasia of aorta and vessels Transposition of great vessels
3. ANOMALIES IN THE SIZE AND POSITION OF THE HEART	Transposition of heart chambers Dextrocardia Congenital hypertrophy Ectocardia	

Regarding the relative frequency of the different lesions valuable information is contained in the statistical table of Abbott,[1] based upon 631 cases. The following figures are abstracted from it:

TABLE 85.—FREQUENCY OF DIFFERENT FORMS OF CONGENITAL CARDIAC DISEASE

Variety	No. of cases
Displacement of the heart	30
Partial defect of the interauricular septum	237
Partial defect of the interventricular septum	196
Complete defect of one or more septa, or of both	34
Anomalies in the origin of the arteries	184
Pulmonary stenosis or atresia	150
Aortic stenosis or atresia	21
Tricuspid stenosis or atresia	20
Mitral stenosis or atresia	9
Patulous ductus arteriosus	193
Stenosis (coarctation) of the aorta	102
Hypoplasia of the aorta	46

The tendency is for more than one lesion to exist at a time rather than singly, and very varied combinations are met with. In the list given the very great majority are combined with other cardiac defects. Most of the lesions mentioned are uncommon and present no diagnostic symptoms by which they can be recognized during life. Those of most importance are defects in the interauricular septum; defects in the interventricular septum; stenosis of the pulmonary orifice; patulous ductus arteriosus.

1. Congenital Defect in the Interauricular Septum.—This is a relatively common lesion, but usually of no great clinical importance. In fact, a slightly patulous condition of the foramen ovale was found in 44 per cent. of 800 autopsies reported by Wallmann.[2] Fisher[3] observed slight patency present in over 25 per cent. of post-mortems on adult subjects. Generally the opening is small, and in such cases, if occurring alone, it can hardly be called pathological. When large enough to constitute an abnormal condition, it is present oftenest in combination with other congenital cardiac defects. In Abott's[1] 631 cases of congenital heart-disease there were 89 of persistence of the foramen ovale of a size which could be called pathological, and in 18 of these it was the only lesion present. Much more rarely the lower portion of the interauricular septum may exhibit defective formation, showing one or more apertures, or the septum may even be entirely wanting.

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[1] Osler and McCrae, Modern Med., 1915, 342.

[2] Prager Vierteljahrschrift f. d. prakt. Heilk., 1859, XVI, II, 20.

[3] Rep. Brit. Soc. Dis. in Child., 1902, II, 263.

2. Congenital Defect in the Interventricular Septum.—This is among the most frequent of congenital cardiac lesions. It occurs usually in association with anomalies in the condition of the large vessels or orifices; and probably the most common condition with clinical manifestations seen in congenital cardiac disease is the combination of this with pulmonary stenosis, and often with patulous ductus arteriosus. It is found alone in a very much smaller number. The opening is situated at the upper part of the septum in the large majority of cases and may be very small, but is generally big enough to admit the tip of the little finger. Sometimes it is larger than this, and even the entire septum may be absent, thus producing a heart with two auricles and one ventricle; the *cor triloculare*. If the auricular septum is also absent the *cor biloculare* results.

3. Stenosis or Atresia of the Pulmonary Ostium.—This is one of the most common of congenital lesions of the heart and the most important of those which permit of the continuance of life. It was present in 150 of Abbott's[2] series of 631 cases. Peacock[3] reported atresia or stenosis in 119 of 181 cases of congenital heart-disease; *i.e.* 65.75 per cent.; and in patients surviving the age of 12 years, it was observed 38 times in 45 cases; *i.e.*, 84.44 per cent. Hochsinger[4] estimated it at about 60 per cent. of all cases. The narrowing of the pulmonary ostium may be of various degrees. Complete atresia is less common than stenosis. Vierordt[5] found 83 collected cases of stenosis and 24 of atresia; a ratio of 3.46 : 1. The contraction is sometimes in the ostium alone, but in the large majority of cases the conus arteriosus is also involved. The semilunar valves may be more or less movable or may be stiffened into entire immobility. Pulmonary stenosis generally occurs in combination with other congential cardiac diseases; in fact is a cause of them in many instances, they existing as compensatory lesions to admit of the better passage of blood from the right side of the heart. Consequently stenosis is present in combination with defect of the ventricular septum more frequently than it occurs alone. In Vierordt's[6] 83 cases of stenosis, 71 (85.5 per cent.) had also septum-defect; and in the 24 cases of atresia, 14 (58.3 per cent.) had this lesion. In 192 cases of pulmonary stenosis collected from medical literature Rauchfuss[7] noted 171 with combined defect of the ventricular septum, and only 21 without this. This association is especially true of patients over 1 year of age, inasmuch as stenosis occurring alone does not usually permit of a long continuance of life. A patulous foramen ovale is a very frequent attendant upon pulmonary stenosis. Küssmaul[8] reported the foramen open in 39 out of 53 cases (73.6 per cent.). The ductus arteriosus is also patulous in many instances

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[1] *Loc. cit.*, 356.
[2] *Loc. cit.*, 389.
[3] Malformations of the Heart, 1866, 193.
[4] Pfaundler und Schlossmann Handb. d. Kinderh., 1910, III, 506.
[5] Nothnagel, Spec. Pathol. u. Therap., 1901, XV, 2, 77.
[6] *Loc. cit.*
[7] Gerhardt Handb. d. Kinderkr., 1878, IV, 1, 69.
[8] Zeitsch. f. rationale Medecin, 1865, XXVI, 161.

According to Küssmaul this was the case in 9 out of 39 cases (23.1 per cent.) of stenosis and in 14 out of 17 (82.53 per cent.) cases of atresia.

4. **Patulous Ductus Arteriosus.**—This, like patulous foramen ovale, is not a malformation but the persistence of a fetal condition normally present at birth. It is common in association with other lesions, especially pulmonary stenosis and perforate ventricular septum; but is infrequent alone. Vierordt[1] collected but 26 uncomplicated cases from medical literature. Abbott's[2] list contains 64 instances. It is usually a secondary lesion, oftenest to those of the septum and pulmonary artery, and is then compensatory, offering a means for the unoxygenated blood to reach the lungs. In other cases it is the result of early pulmonary atelectasis. When of small lumen, as of a size sufficient to admit only a probe, it has no clinical significance.

Other Congenital Disorders of the Heart.—These are either less common or have no special clinical significance. Congenital *disease of the tricuspid orifice* is not common and rarely occurs except in combination with other cardiac lesions; and that of the *mitral orifice* is still rarer. J. Hess[3] was able to collect but 8 reported cases of uncomplicated atresia of the tricuspid orifice, to which he added another. Stenosis or atresia of the *aortic ostium* likewise is infrequent. It may or may not be associated with perforate septum or with patulous foramen ovale. In rare instances a subaortic stenosis has been observed (Thursfield and Scott).[4] There may be an *anomalous origin of the vessels* leaving the heart. This is a not uncommon condition. Various forms may be seen, as when both vessels arise from one ventricle, or the aorta from both; or both vessels from one trunk, or an incomplete separation of the aorta and pulmonary artery exists. One of the most interesting forms is complete transposition in which the pulmonary artery arises from the left ventricle and the aorta from the right (Fig. 305). This occurred in 43 of Abbott's series. All these anomalies are always associated with other cardiac malformations. Congenital *narrowness* or *dilatation of the pulmonary artery* may occur, the former oftenest in combination with stenosis of the pulmonary orifice. Dilatation of the pulmonary orifice is uncommon occurring alone, but often seen in combination with other lesions. It is associated oftenest with defect in the interventricular septum or with patulous ductus arteriosus. A not uncommon condition is *stenosis of the isthmus of the aorta* near the origin of the ductus arteriosus. This is of two types, as described by Bonnet;[5] the infantile, in which there is a diffuse narrowing of the isthmus of the aorta, slight or severe, which is generally accompanied by a patulous ductus arteriosus; and the adult type, which is produced in the process of closing of the ductus, and results in a narrow constriction of the aorta between the ductus and the left subclavian artery. The infantile type is associated with numerous other cardiac malformations. It is not often encountered after the period of infancy. The other more frequent form, called also *coarctation of the aorta* is accompanied later by extensive development of the collateral circulation. A general *hypoplasia of the aorta and arterial system,* with smallness of the heart, is sometimes seen. Finally in rare cases a *congenital cardiac hypertrophy* may occur, or the apex of the heart points to the right (*dextrocardia*); or its *chambers are transposed*, often in combination with transposition of the abdominal viscera; or the heart may project through an opening in the diaphragm into the abdominal cavity, or from the body through an abnormal aperture in the chest-wall (*ectocardia; ectopia cordis*). This last is often associated with a congenital absence of the pericardium.

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[1] *Loc. cit.*, 160.

[2] *Loc. cit.*, 410.

[3] Amer. Jour. Dis. Child., 1917, XIII, 167.

[4] Brit. Jour. Child. Dis., 1913, X, 104.

[5] Rev. de méd., 1903, XXIII, 108.

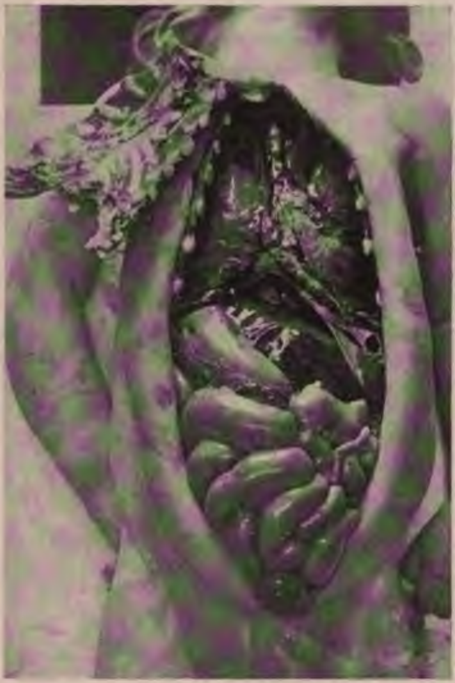

FIG. 305.—TRANSPOSITION OF THE GREAT VESSELS AND OF VISCERA IN A CASE OF CONGENITAL CARDIAC DISEASE IN A MALE INFANT OF 8 MONTHS.

Cavæ emptied into the left auricle, pulmonary veins into the right. Aorta arose from right ventricle and descended on right side of the spinal column. Pulmonary artery arose from left ventricle, small, orifice stenosed. Upper part of ventricular septum deficient. Right lung 2 lobes, left lung 3. Stomach, liver, spleen, and cecum transposed (*Griffith, Univ. Med. Magaz.*, 1899, *Aug.*)

General Symptomatology.—Although there is great variation possible in the symptoms, in general there are certain manifestations which, when present, render the existence of congenital cardiac disease very probable. Among these are especially the following:

Cyanosis.—This is observed usually soon after birth and may be very intense and distributed over the whole surface, including the mucous membranes especially of the mouth. In other cases, although appearing equally early, the cyanosis is limited to the fingers, toes, lips, nose, cheeks, and ears; may be evident only after crying, or, in older children, running or other exercise, or conditions of excitement; or may appear especially when the child is suffering from some pulmonary disorder. In still other instances cyanosis does not develop until the child is some months or even years of age, and then may be general or limited; continuous or intermittent. In well-marked cases the skin is of a bluish-red or of a slate color and the mucous membrane of the mouth is purple-red (Figs. 306, 307, 308). In long-continued cases dilatation of the veins and of the cutaneous capillaries occurs and the retinal vessels may be dilated and tortuous.

The cause of the cyanosis has been much disputed. The view that it is due to a mixture of arterial and venous blood has largely given place to the opinion that it depends upon an imperfect oxygenation of blood from a passive congestion in the venous system, or from the inability of a sufficient amount of venous blood to reach the lungs. The question is, however, by no means settled, and cases in evidence and in contravention are advanced upon each side. The absence of cyanosis by no means proves the absence of congenital cardiac disease. In fact this symptom is not observed in many cases even with severe cardiac malformation. The nature of the malformation is the deciding element in the production of the symptom, pulmonary stenosis occupying perhaps the chief position here. Transposition of the great vessels is a less frequent cause. On the other hand, the presence of congenital cyanosis is not of itself proof that congenital malformation of the heart exists, since it may be well marked in cases of congenital atelectasis and other conditions.

Blood.—With the cyanosis there are characteristic alterations of the blood. The amount of carbonic dioxide present is augmented and the number of red blood-cells is decidedly above normal, reaching frequently as high as 7,000,000 or 8,000,000 to the cubic millimetre or sometimes even over this. Bach[1] reported a case with 11,400,000 red blood-cells to the cubic millimetre and a hemoglobin percentage of 150; and still larger numbers of corpuscles have been recorded. The percentage of hemoglobin is usually proportionately increased.

Clubbing of the Fingers.—This is a very characteristic symptom seen in severe cases. The whole terminal phalanx of the finger or toe is enlarged (see illustration); either the soft parts only, or there is an osteoperiostitis as well. A similar condition although without cyanosis may be seen in chronic pulmonary diseases. (See also p. 427, Fig. 379.)

Other Symptoms.—Among other symptoms observed is dyspnea, which, with increased rapidity of respiration, is present more or less in all cases of cyanosis. It may be slight or occur only on exertion; or there may be in bad cases in infants and young children severe suffocative or anginoid attacks, during which the cyanosis becomes intense and death seems imminent. The attacks may be accompanied by unconsciousness or by epileptiform convulsions. Hemorrhages from the mouth or nose occasionally occur. Edema is not a common symptom except as failure of compensation becomes marked. Palpitation is sometimes a troublesome symptom. The temperature of the body is usually subnormal, the extremities cold, and the patient complains of chilliness. The pulse is generally rapid, exceptionally is slow, and palpitation is sometimes annoying. Bodily and mental development are retarded in well-marked cases, the general nutrition suffers, and a condition of infantilism may follow.

Cardiac Examination—Reserving this chiefly for more detailed consideration under the diagnosis of the different forms of malformation, it may be said here that in general there is found a murmur, unusually loud and of harsh quality, systolic, and often widely diffused. With this is often a precordial thrill. An accentuated pulmonary second sound is common. In contradistinction to the loudness of the murmur is the fact that there is little cardiac enlargement, at least early in the case. Later

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[1] Arch. f. Kinderheilk., 1909, L, 31.

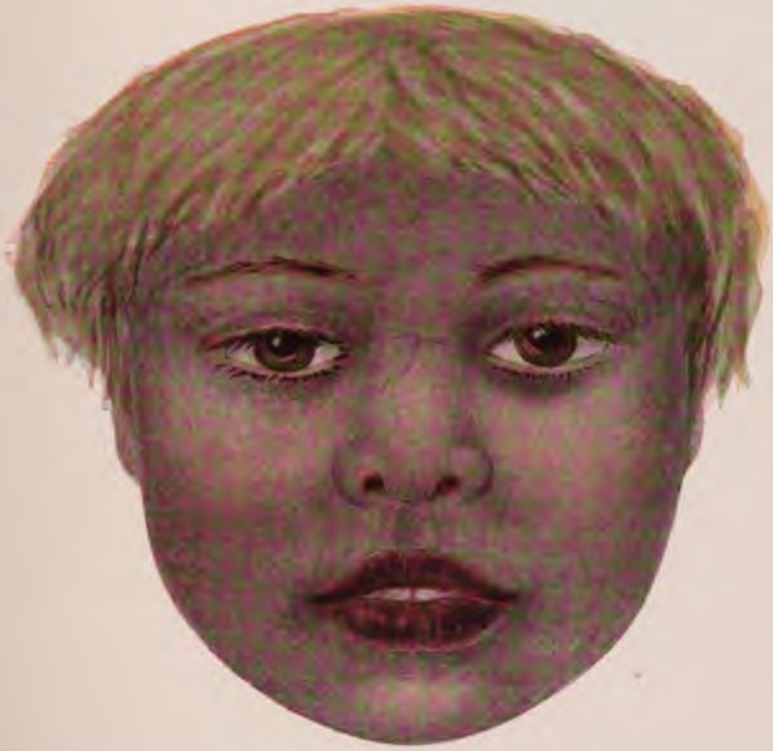
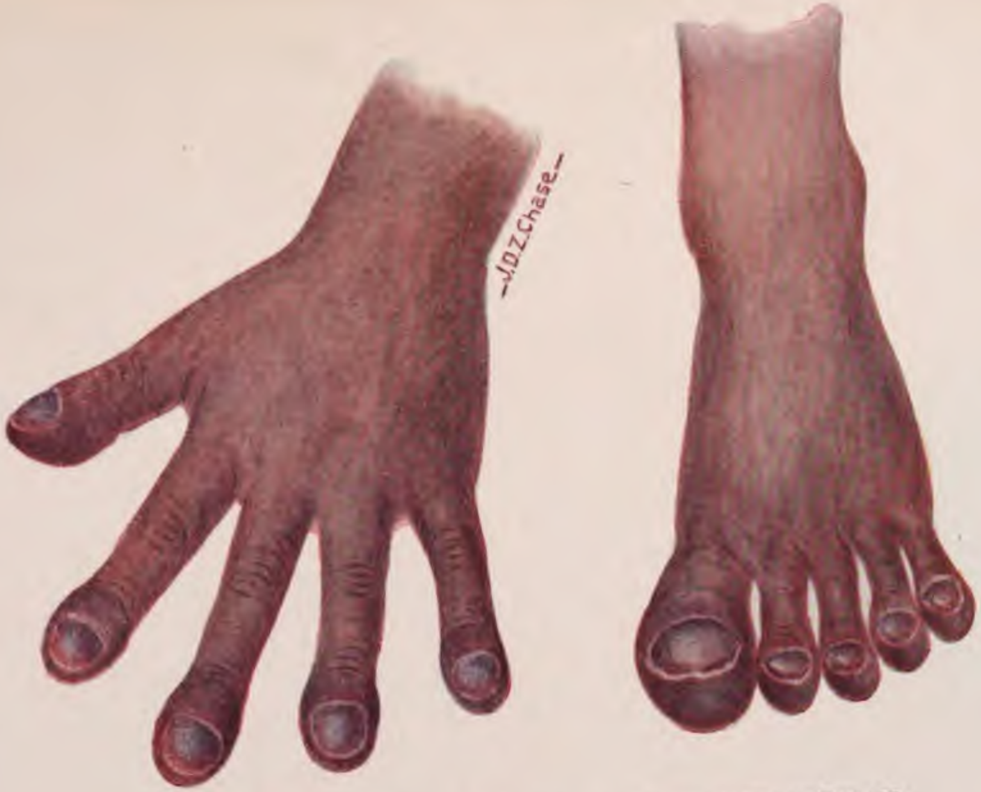


FIG. 306.—CONGENITAL DISEASE OF THE HEART.

Boy, aged 3 years. Intense, wide-spread cyanosis; attacks of syncope and severe dyspnea. No murmur. Red blood-cells 9,040,000; hemoglobin 120 per cent. *X*-ray showed decided enlargement of the heart to the left. Condition presumably anomalous origin of the great vessels.



FIGS. 307 and 308.—CLUBBING OF FINGERS AND TOES IN CONGENITAL HEART DISEASE.
(Same case as in Fig. 306.)

in the course enlargement occurs, limited chiefly to the right side of the heart.

Diagnosis.—The diagnosis in general rests upon the data already given. A patient in the first 2 or 3 years of life, with cyanosis, thrill, a loud murmur, and only moderate enlargement of the right side of the heart, usually not in proportion to the cardiac symptoms and physical signs, is probably suffering from congenital cardiac disease. As a rule the murmur is loud, rough, and sometimes musical, and heard best at the mid-sternum or to its left; and the apex beat is weak or at least not increased in strength. Yet the diagnosis is often difficult and sometimes impossible; especially as grave cardiac lesions may exist without in some cases producing any murmur whatever, or in other cases any cyanosis. Postnatal lesions depending upon endocarditis exhibit the symptoms and physical signs characteristic of the variety chancing to be present; and the position of maximum intensity and nature of diffusion of the murmur vary with the lesion and are very different from those observed in the congenital cases. Cyanosis in acquired disease of the heart is a later symptom, edema usually developing first. There is generally, too, the history of an antecedent rheumatism or some other cause.

The diagnosis of congenital murmurs from those of an accidental nature is sometimes difficult. The latter are either functional, and then of a softer, less diffused nature; or are dependent upon anemia, and then are transmitted into the vessels of the neck and are attended by evidences of anemia but not of enlargement of the heart. The cyanosis of congenital heart-disease is to be distinguished likewise from that due to other causes; notably atelectasis in infants; and later from the cyanosis with polycythemias and enlargement of the spleen described by Vaquez,[1] and which is known by his name. (See also p. 451.)

The diagnosis of the different forms of congenital disease of the heart from each other is, as a rule, a matter of great difficulty and uncertainty; and in most cases can be made only provisionally. The variety of combinations which may occur is great, and there is moreover no lesion or combination of lesions which presents always the same physical signs, the exceptions to what in theory should be expected being very numerous and contradictory. Further, lesions may be found at autopsy in patients who have exhibited no positive evidence of cardiac disease during life. Nevertheless, the nature of the lesion present has many times been diagnosed correctly, as proven later by autopsy, and the effort should always be made; especially as the prognosis varies with the form of lesion present. As far as our knowledge extends the following symptoms and physical signs are to be expected:

Defect in the Interauricular Septum.—It is questionable whether patulous foramen ovale or other defect of the septum is productive of any symptoms, especially when occurring alone. When associated circumstances greatly disturb the relative intracardial pressure in the two auricles, cyanosis may develop and a diastolic murmur over the upper part of the sternum may be heard in some instances. Such disturbing conditions are, however, usually due to the presence of other congenital cardiac malformations, and these obscure the diagnosis of inter-auricular defects.

Defect in the Interventricular Septum.—Perhaps of all the varieties this gives the most characteristic symptoms and physical signs; yet it is possible for it to exist without any evidence whatever during life.

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[1] Le bull. méd., 1892, VI, 849.

Occurring alone it has been described especially by Roger[1] and is often named for him, "Maladie de Roger." In this form cyanosis and dyspnea may be absent or present, varying with the case and depending on factors not well understood. In Abbott's[2] series cyanosis was observed in half of the cases. When pulmonary stenosis exists in combination with septum-defect cyanosis is usually present. The absence of cyanosis is, with few exceptions, an indication that there is no accompanying pulmonary stenosis. Examination of the heart generally reveals a loud systolic murmur over the whole precordium, with maximum intensity at the mid-sternum or in the 3d interspace just to the left of the sternum. The murmur is also heard at the apex, although generally less loud here, and is widely diffused, being audible even in the back, but in uncomplicated cases not conducted along the large vessels. All the valve sounds are audible. The murmur is produced in cases of simple perforate septum by the blood passing from the left ventricle into the right. This necessarily increases the pressure in the right side, normally less than on the left, and the pulmonary second sound is consequently accentuated to a moderate degree. In cases combined with pulmonary atresia or well-developed stenosis the blood passes in the other direction; *i.e.*, through the perforate septum from the right side to the left, as this is often the only sufficient method of exit; and in some such cases, the murmur, being produced by whirls of blood just beneath the opening of the aorta, may be transmitted along this vessel into the carotid and subclavian arteries.

The loudness, or even the presence, of the murmur is not in proportion to the size of the defect in the septum. Even large openings may sometimes be unattended by murmur, and if the septum is completely wanting no murmur will be heard. A distinct systolic thrill is sometimes felt, but is not a necessary accompaniment. If the case is long-continued, enlargement of the right side of the heart will develop, but in earlier cases is not discovered, especially when this lesion occurs alone.

Uncomplicated defect of the ventricular septum is to be distinguished from pulmonary stenosis by the lower situation of the murmur; its wider diffusion; the more frequent absence of cyanosis in uncomplicated cases; and the presence of valvular sounds when cyanosis does exist. Very important in uncomplicated cases of each condition is the auscultation of the pulmonary second sound. In perforate septum the sound is accentuated; in pulmonary stenosis it is weaker than normal. In the numerous cases in which septum defect is combined with pulmonary stenosis the diagnosis can be made only by a careful consideration of all the symptoms. Auscultation alone is hardly sufficient.

The diagnosis of septum defect from early acquired mitral regurgitation is often of great difficulty and sometimes impossible. However, in mitral disease the symptoms and signs develop generally after the 1st year of life; there is enlargement of the left ventricle; the murmur is heard usually loudest at the apex; the first sound of the heart here is obscured; there is usually no thrill; edema generally develops before cyanosis; and there is not the decided retardation of general development.

Stenosis or Atresia of the Pulmonary Orifice.—This may exist uncomplicated, or much more frequently combined with other lesions. Cyanosis is present in the majority of cases, usually seen immediately or soon after birth. Sometimes its appearance is delayed, and not infrequently it is replaced by pallor. With the cyanosis are the other symptoms usually accompanying it, namely polycythemia, clubbing of the fingers, palpitation, dyspnea, suffocative attacks, etc. Enlargement of the right side of the heart is a valuable diagnostic sign often well-marked in older children, or earlier in cases of uncomplicated pulmonary stenosis. Frequently the enlargement of this chamber seems but slight, inasmuch as the tension on the ventricular walls produced by the stenosis is relieved by the so common presence of septum defect. A systolic thrill is sometimes felt, but is oftener absent.

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[1] Bull. de l'acad. de méd., 1879, Ser. 2, VIII, 1074.
[2] *Loc. cit.*

Auscultation usually reveals a systolic murmur with maximum intensity over the pulmonary cartilage to the left of the sternum or in the 2d left interspace. It is conducted well to the left, but its intensity diminishes rapidly toward the right. Cases may occur, however, in which no such murmur is audible. This is true, for instance, of complete uncomplicated atresia of the pulmonary orifice, or when the opening is very small. In many cases the exact localization of the murmur is rendered most difficult by the presence of other murmurs, especially that of defect of the septum. When the two are associated the combined murmur is loudest in the 2d or 3d interspace to the left of the sternum, and may be conducted along the carotids. Sometimes in such cases it is possible to observe that there are two areas in which the murmurs are of different quality and pitch.

The condition of the pulmonary second sound is of great importance. In uncomplicated pulmonary stenosis, at least in young subjects, the sound is always weaker than normal. This is the necessary result of the small amount of blood which enters the pulmonary artery and the consequent diminished diastolic pressure there. Later in life the weakness of this sound may be removed through decided hypertrophy of the right ventricle; or in complicated cases the tension within the right ventricle may be increased by the existence of a septum defect which allows the greater power of the left ventricle to exert a direct influence. In either of these ways the second sound may be made of normal strength. When, however, there exists a patulous ductus arteriosus and the pulmonary leaflets are freely movable the pulmonary second sound is very much accentuated owing to the increased direct pressure in the pulmonary artery which is the natural result of communication with the aortic system.

In brief, the diagnosis of pulmonary stenosis and of atresia rests upon the presence of decided cyanosis and accompanying symptoms, and, in the case of stenosis, of a systolic murmur to the left of the sternum in the 2d or 3d interspace. A feeble pulmonary second sound indicates simple stenosis; a normal pulmonary second sound an accompanying septum defect, and an accentuated one the presence with these of a patulous ductus arteriosus. When there is little if any hypertrophy of the right ventricle it is probable that a communication exists between the auricles or the ventricles respectively. The presence of pallor instead of cyanosis, combined with the murmur at the pulmonary orifice, may readily lead to the mistaken diagnosis of a persistent, severe anemia with a murmur of functional origin. This is, according to Hochsinger[1] particularly true of moderate stenosis of the conus arteriosus, in which murmurs may come and go from time to time, exactly as in the case of anemic murmurs. Very important, too, in the differentiation between simple and complicated pulmonary stenosis is the fact that very few cases of the former, if at all well marked, are encountered after the end of the 1st year of life.

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[1] Die Auscultation des kindlichen Herzes, 1890, 178.

Patulous Ductus Arteriosus.—In the very exceptional cases of uncomplicated open ductus there are no characteristic symptoms. Cyanosis is usually absent; hypertrophy of the right side of the heart is frequent and sometimes that of the left side also; there has been found a small area of percussion-dullness in the 1st and 2d interspaces to the left of the sternum; there is a loud systolic murmur heard over the pulmonary cartilage and conducted into the vessels of the neck, but not downward, and often uniting with a diastolic murmur and producing a continuous rumble ("humming-top murmur"). The diastolic murmur depends upon insufficiency of the pulmonary orifice. There is often a systolic thrill in the 2d left interspace and the pulmonary second sound is much accentuated. This accentuation is a diagnostic sign of very great importance and is the natural result of the diastolic action upon the pulmonary leaflets of the combined aortic and pulmonic pressure. An enlargement of first the right and then the left side of the heart is considered by Hochsinger[1] of especial diagnostic significance, since no other congenital cardiac lesion recognizable during life will produce this.

When patulous ductus arteriosus occurs in combination with other lesions the symptoms observed are largely those of these latter, and those of the former may be modified. If there is atresia of the pulmonary artery, a murmur is usually not audible, at least at the ductus arteriosus, as apparently both pulmonary artery and aorta must supply blood in order to produce it. The coexistence of pulmonary stenosis and patulous ductus will produce a murmur the localization and nature of which possess the character of the murmur of each of these, which cannot then be separated one from the other. The existence of the combination is suggested by the presence of cyanosis, the marked accentuation of the pulmonary second sound usually present, the harshness and loudness of the murmur, and the existence of thrill.

Other Congenital Lesions.—Other lesions possible in congenital heart-disease are for the most part either of very uncommon occurrence, or not susceptible of clinical recognition. Anomalous origin of the pulmonary artery and aorta, or even transposition of these, although a comparatively frequent form of disorder, exhibits no certain symptoms. Suggestive of transposition is the complex of symptoms pointed out by Hochsinger,[2] viz., cyanosis with much accentuated pulmonary second-sound and enlargement of the heart; but without any murmurs whatever. Dilatation of the pulmonary artery produces of itself no diagnostic symptoms. That of the orifice gives rise to a diastolic murmur. Congenital narrowness of the aortic orifice is not common. Its symptoms would probably not differ from those of the lesion acquired later in life; characterized especially by enlargement of the left ventricle, displacement of the apex-beat, and a systolic murmur carried into the vessels of the neck; but the duration of life is so short that a diagnosis could hardly be made. Atresia of the aortic orifice causes no murmur whatever even when a patulous ductus arteriosus is present. In such cases the blood must reach the aorta by way of a perforate septum taking the blood from the left ventricle, and a patulous ductus arteriosus transmitting it from the pulmonary artery. There is intense congestion of the lungs, and life continues for only a few weeks. Stenosis of the aorta in the neighborhood of the entrance of the ductus arteriosus (coarctation of the aorta; stenosis of the isthmus) is seen more frequently. It may exhibit no evidences of its presence until childhood is passed; or there may be early symptoms consisting of a systolic murmur at the upper part of the sternum and to its right, extending to the jugulum, where pulsation can be detected, and conducted into the vessels of the neck. The left ventricle is hypertrophied. The pulse in the vessels of the lower extremities is weaker than in those of the upper and of the neck. In subjects reaching adult life with considerable stenosis of the isthmus there develops a very distinct establishment of collateral circulation through the upper intercostal and internal mammary arteries connecting with the epigastric arteries and lower intercostals. The surface of the thorax and abdomen may show the branching, much dilated vessels. This is the result of the effort to furnish blood to regions below the situation of the stenosis. Congenital hypoplasia of the general aortic system has no typical cardiac symptoms, except that early in life the heart is abnormally small. There is some hypertrophy later in a considerable number of cases. The general symptoms are those of anemia and debility with retarded development of the genital organs. (See Chlorosis, p. 460.) Unassociated disease of the tricuspid or mitral valve is rare. Although in uncomplicated cases the symptoms should be those of the acquired condition, inasmuch as these lesions nearly always occur only in combination with others, the differential diagnosis is usually impossible.

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[1] *Loc. cit.*, 168

[2] *Loc. cit.*, 155.

A brief review of the principal diagnostic features differentiating some of the various lesions follows:

1. *Cyanosis decided, without murmur,* suggests transposition of the large vessels without other complicating lesions. Corroborative of this is accentuated pulmonary second sound. The cyanosis may, however depend upon other cardiac malformations or upon other diseases. Exceptionally cyanosis without murmur may be due to atresia or a very great stenosis of the pulmonary orifice.

2. *Cyanosis with systolic murmur in the 2d left interspace* suggests pulmonary stenosis. The stenosis is uncomplicated or only with patulous foramen ovale if the pulmonary second sound is feeble. It is combined with septum defect, if the pulmonary second sound is normal or somewhat accentuated. If the second sound is much accentuated, it is probable that patulous ductus arteriosus is also present.

3. *Systolic murmur loudest over mid-sternum or in the 3d left interspace, without cyanosis,* suggests probable simple septum defect. The murmur is then not conducted along the large vessels.

4. *Systolic murmur very loud and harsh over the 2d interspace or upper part of the sternum and carried into the vessels of neck, with great accentuation of the pulmonary second sound,* indicates the presence of patulous ductus arteriosus with pulmonary stenosis. The existence of patulous ductus arteriosus is corroborated by enlargement of the left side of the heart as well as of the right side. Either patulous foramen ovale or ventricular septum defect will also be present. The absence of cyanosis might indicate that the patulous ductus was an isolated lesion.

5. *Systolic murmur over upper part of sternum and to its right and carried into the vessels of the neck, with hypertrophy of the left ventricle,* suggests stenosis of the isthmus of the aorta. It is corroborated later in life by visible collateral circulation.

6. *Diastolic murmur* usually suggests pulmonary insufficiency; sometimes the existence of patulous foramen ovale.

As earlier stated, however, owing to the great variation of the symptoms observed and the many possible combinations, the diagnosis of the forms of congenital heart disease present can seldom be more than provisional.

Prognosis.—In general the prognosis of congenital cardiac heart disease is unfavorable. More than half of the cases die during the first 5 years of life. Very many live but a few days or weeks, the nature of the lesion preventing a longer existence. Others die in the early years, either through the development of lack of compensation, or from the occurrence of complicating diseases. On the other hand, some reach adult life either with manifest symptoms or sometimes without any of moment. It is also possible for infants with evidences of congenital cardiac disease to lose their symptoms and physical signs later, either temporarily or permanently.

As regards the relationship of *complicating conditions*, there is a tendency to the development of pulmonary disease, and such an occurrence is always serious. Pertussis is borne very badly. Rachitis influences the patient unfavorably as do gastroenteric affections. Sometimes endocarditis is added to the cardiac lesions already present. Tuberculosis is liable to develop in those passing the years of childhood. The acute exanthemata are generally fairly well tolerated.

As far as the indications from the symptoms go, the prognosis seems to be to a large extent unfavorable in proportion to the degree of cyanosis; and of the dyspnea, palpitation and the like which are liable to accompany this. Infants born with intense cyanosis generally die very soon; those without it, or who soon lose it if present at first, give the best prognosis, as a rule.

Viewing the prognosis from the standpoint of the *individual lesion* the following analysis may be made:

Defect in the interauricular septum, uncomplicated or producing no symptoms, affects in no way the duration of life. If other lesions are present or develop later, the septum defect may affect life by producing overfilling of the right auricle.

Defect in the interventricular septum, if uncomplicated, often permits of life into adult years. It is, however, prone to be complicated at this period or earlier by endocarditis affecting the neighborhood of the opening or other part; and this, or the presence of other lesions of a congenital nature, may modify the prognosis and shorten life.

Pulmonary atresia, occurring alone, usually causes death in a few weeks. If compensated by perforate septum with patulous ductus arteriosus life is longer. Yet the average duration of life in all cases of atresia, according to the calculations of Vierordt,[1] based on Rauchfuss' statistics, is only 3.27 years.

Pulmonary stenosis gives a better prognosis; yet fully 50 per cent. of the cases die before the age of 10 years, and 75 per cent. of them before that of 15 years. Vierordt[2] reckons the average duration of life of pulmonary stenosis, based on Moussous' cases, as 12 to 13 years; or, based on Rauchfuss' collection, as 9.36 years; and cases without compensatory septum defect are still less favorable. On the other hand, many patients with pulmonary stenosis live to adolescence or even adult life. An unfavorable termination is attended by increasing loss of cardiac compensation, with general passive congestion and edema.

Patulous ductus arteriosus occurring alone has on the whole a not very unfavorable influence upon the duration of life. Of the 26 cases collected by Vierordt[1] over half lived to adolescence or adult life. In fatal cases there is progressive failure of compensation, perhaps due to a secondary endocarditis. A patulous ductus arteriosus associated with pulmonary stenosis acts rather as a compensatory lesion.

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[1] *Loc. cit.*, 83.

[2] *Loc. cit.*, 104.

Anomalous origin of the great vessels gives a prognosis varying with the exact nature of the condition. Some of these do not permit of life at all, while in others adult life is exceptionally attained. Cases of transposition of the vessels seldom live beyond the 2d year. In 75 cases collected by Taruffi[2] only 9 were over 5 years of age.

Stenosis of the aortic ostium presents a very unfavorable prognosis, most cases dying in the 1st week; only 1 of 33 cases collected by Rauchfuss[3] passing the 1st month of life.

Stenosis of the isthmus of the aorta at the position of the ductus arteriosus gives a much better prognosis. The course is chronic and the patient frequently reaches adult life with but few symptoms. The fatal ending comes probably from heart-strain during middle age, with evidences of failing cardiac compensation.

Treatment.—This can be but symptomatic, while the nature of the lesion often renders even this valueless. The greatest care to sustain and advance the state of the nutrition; the guarding against chilling and especially against over-exertion; and, in general, exact attention to hygienic and dietetic management are the only means open to us. Respiratory diseases are especially to be prevented, since patients with congenital cardiac disease are particularly liable to contract them and bear them very badly. In some cases the continued administration of nitro-glycerine appears to be of benefit, probably through relieving the strain on the heart by dilating the peripheral vessels. In others, subject to sudden attacks of intense cyanosis and cardiac pain, the hypodermic injection of nitroglycerine or of morphine or the inhalation of nitrite of amyl is of value during the attack. Inhalations of oxygen have been tried to relieve cyanosis. If syncope occurs, hypodermic injections of camphor or caffeine may be given.

CHAPTER III

PERICARDITIS; ENDOCARDITIS; MYOCARDITIS

ACUTE PERICARDITIS

Etiology.—This disorder is not common in infancy and early childhood, but after this period is at least as frequent as in adult life. Probably more cases of pericarditis occur in later childhood than at any other time. Cnopf[4] found it in 7 of 130 autopsies in children, and Babinsky[5] 66 instances of it in 4500 sick children. Of these 66, 20 occurred in the 1st year; 37 in the first 5 years; 16 from 5 to 10 years, and 13 from 10 to 14 years. The disease is never primary, except perhaps as the first or only manifestation of rheumatism, and in many such cases it is very probable that a rheumatic affection elsewhere in the body has been unrecognized. Among other causes are the various acute infectious diseases, such as typhoid fever, scarlet fever, diphtheria, measles, varicella, smallpox, and erysipelas. To be mentioned also are nephritis, tuberculosis, and septic processes in various parts of the body. Inflammation may spread to the pericardium by extension in cases of pneumonia and pleurisy, and in inflammatory disease of the abdominal structures, bronchial glands or the bony framework of the thorax. Trauma constitutes a local cause in some instances.

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[1] *Loc. cit.*, 164.

[2] Mem. de la Soc. med.-chir. di Bologna, VIII, Fasc. 1. Ref. Vierordt, *loc. cit.*, 128.

[3] *Loc. cit.*, 134.

[4] Münch. med. Woch., 1889, XXXVI. 357.

[5] Berl. klin. Woch., 1898, XXXV, 1053.

The relative frequency of these agents depends upon the age of the patient. Rheumatism is rarely a cause under the age of 5 years since that disease is so seldom seen at this period. In later childhood it is by far the most frequent source. It happens oftener than in adult life that a rheumatic pericarditis, generally combined with endocarditis, develops without or before any arthritic involvement, very often associated in some way with chorea. Septic conditions and especially pneumonia are the most common forerunners of pericarditis in infancy and early childhood. Tuberculosis is an infrequent cause in the first 2 years of life, and after this time is less often operative than is the case with adults. The other infectious disorders are factors chiefly in later childhood. Extension of inflammation from neighboring diseased tissues may occur at any period of early life. Here may be mentioned inflammation of the thymus or lymphatic glands, the neighboring bony structures, the esophagus or the peritoneal cavity.

Pathological Anatomy.—The lesions do not differ materially from those seen in adults, except for the greater frequency of purulent exudate. Either the visceral or the parietal layer or both may be involved. The nature of the lesions is entirely analogous to that observed in pleurisy (p. 101). In *plastic* (dry) pericarditis the pericardium is injected and dry, or, in severer cases, is covered by a more or less thick deposit of fibrin containing pus-cells. In the *sero-fibrinous* form there is an effusion of serum containing cellular elements and flakes of fibrin. The fluid may be clear or somewhat turbid. Sometimes extravasation of red blood-cells gives a sanguinolent appearance to it. Cellular infiltration and a varying degree of degeneration of the cardiac muscle may be present in severe cases. The amount of fluid is usually but small, but sometimes it is so large that errors in diagnosis are readily made. In *purulent* pericarditis the conditions are as in the serofibrinous form except for the presence of large numbers of pus-cells. The exudate may be purulent from the beginning or become so only later. Adhesions may develop between the layers of the pericardium in all of the forms of pericarditis, although not often in the suppurative cases; and these may remain after recovery and sometimes occasion complete obliteration of the pericardial sac. In fact, complete disappearance of all evidences of pericarditis is uncommon.

The variety of pericarditis found depends upon the nature of the cause and the character of the germs present. Rheumatism produces generally a plastic or serofibrinous inflammation and endocarditis is very frequently combined with it. This is the form usually seen after the age of 5 years. Any of the pyogenic germs may be producers of purulent pericarditis, but by much the most common is the pneumococcus. In Poynton's[1] series of 100 suppurative cases, the large majority were pneumococcic in origin, and 84 per cent. of these occurred before the age of 4 years. An attendant empyema was found in 54 and simple pleurisy or pneumonia in 31. A complicating valvular lesion was present in only 1 instance. Tuberculosis may give rise to any of the forms. Usually there is then a deposit of miliary tubercles on the pericardium; but in the more chronic tuberculous cases there may be seen nodules in the subpericardial tissue.

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[1] Quart. Journ. of Med., 1907–8, I, 225.

The external layer of the pericardium is not infrequently attacked in cases suffering from pleurisy in the region of the mediastinum, and firm adhesions form between the pleura and the pericardium, binding the latter to the chest-wall. Inflammation of the inner lining of the pericardium is then commonly present also. Tuberculosis is a frequent cause of this disorder, which is not to be classified as pericarditis, but better denominated *mediastinitis*. Further is to be distinguished the simple transfusion of fluid into the pericardial sac which is seen in cases of cardiac weakness, anemia and other debilitating conditions, and which is a hydropericardium, not a pericarditis.

Symptoms.—These vary greatly in severity and depend to some extent upon the form of pericarditis present. The condition is readily overlooked because over-shadowed by the malady to which the pericarditis may be secondary, or because the symptoms are so often obscure. Very frequently the diagnosis is made only accidentally. The earliest of the characteristic *physical signs* discovered on cardiac examination is a friction sound of a very rough and superficial nature, best heard over the upper part of the precordial region, but sometimes loudest over the apex. A distinct fremitus can often be felt. Although synchronous with the heart's action it has, in typical cases, little of the character of an endocardial murmur and is usually more circumscribed, as well as more continuous, being heard with both systole and diastole and not transmitted, and varying much in character from time to time. Sometimes it is purely systolic in time. It is intensified by having the patient lean forward or by the pressure of the stethoscope. If effusion take place friction sounds may disappear, unless the pericardial surfaces are loosely bound together in certain locations, still permitting friction to develop here. Even, however, with effusion of considerable size a friction sound may quite commonly still be heard, since the fluid gravitates to a position behind and at the sides of the heart. In other cases disappearance of the friction depends upon a close adhesion of the opposite roughened surfaces of the pericardium. It may sometimes be made more distinct by firm pressure with the stethoscope, or by change of position of the patient, especially by placing him in a sitting position slightly inclined forward. Pericardial friction is encountered chiefly in the cases dependent upon rheumatism. It is uncommon in suppurative cases, Poynton having observed it in only 2 of the 100 cases reported by him.

Another physical sign is increase of the cardiac percussion-dullness dependent upon effusion. A small amount of exudate does not alter the cardiac area perceptibly. Larger amounts may produce the well-known triangular dullness, the apex of this being in the 2d or 3d left interspace; the right side extending below to 1 inch (2.5 cm.) or more beyond the sternum, and the left side reaching beyond the nipple line. Sometimes, however, the dullness does not assume the triangular form, but is more circular and its outline is influenced by gravity, depending on whether the child is reclining or in a sitting position. In the case of very large effusions the dullness may extend well into the left axilla and to the right as far as the nipple. A dullness may be found parallel with the spine upon the left side in cases of large effusion. Dullness to the right of the sternum in the 5th interspace is considered by Rotch[1] to be an important diagnostic sign. It is noteworthy that the apex-beat, when still palpable, is not displaced to the left of the normal position. Such displacement would indicate a complicating cardiac dilatation. With large effusion the apex-beat cannot be felt at all or with great difficulty, and the impulse is diffuse and may occupy a position above the normal position. Having the child lean forward may cause the apex-beat to become again discoverable. Large effusion also alters the character of the heart-sounds, making them weak or scarcely audible; and the precordial region is distinctly too prominent and changes less with respiration. Another sign of importance is that the strength of the radial pulse is out of proportion to that of the apex-beat. The x-ray examination bears out the results obtained by percussion, the shadow showing the triangular or the circular form.

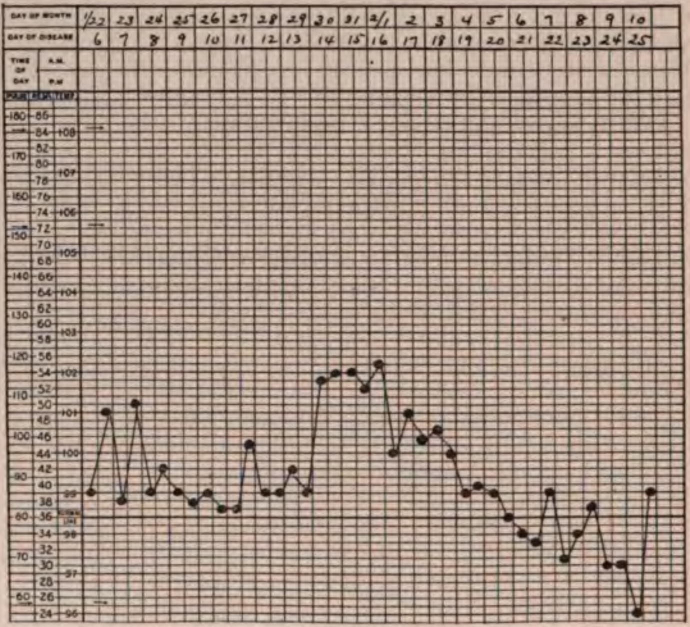

FIG. 309.—PLASTIC PERICARDITIS, FOLLOWED BY ENDOCARDITIS, DILATATION OF THE HEART AND PLEUROPNEUMONIA.

Beckie C., aged 6 years. 5 days previously had pain in the abdomen, and vomited at times after this, insomnia. Entered the Children's Hospital of Philadelphia, Jan. 22. Examination showed typical pericardial friction-sounds over the base of the heart, no dyspnea, general condition good; Jan. 29, apical systolic murmur developed; Feb. 2, very decided dyspnea, evidence of fluid in the left pleural cavity, with bronchial respiration and râles, aspiration in the axillary line gave clear, straw-colored fluid, containing the pneumococcus, signs of increasing mitral regurgitation and of cardiac dilatation appeared. Child grew steadily worse and died Feb. 10.

The *general symptoms* of pericarditis are not characteristic. There is pain and often tenderness in the region of the heart; dyspnea or even orthopnea; palpitation or irregular and rapid cardiac action. Sometimes there is vomiting, cough, and cyanosis. More or less fever usually attends the disease in the early stages, but may be absent. It is of the remittent type in cases of purulent effusion. In patients already suffering from other diseases the possibility of the development of pericarditis is to be considered, when symptoms rapidly appear referable to the heart and not explainable by the preëxisting condition.

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[1] Boston Med. and Surg. Jour., 1878, XXII, 389.

Complications.—That with endocarditis is the most frequent and is very common in cases of rheumatic origin. In those depending upon the pneumococcus pericarditis is often associated with pneumonia or with pleurisy, especially of the left side (Fig. 309). Dilatation of the heart from myocardial changes is a frequent complication in severe cases. Compression of the lung is a natural result of large effusions.

Course and Prognosis.—The *course* varies with the form of the disease. The inflammation may remain plastic throughout, or physical signs of effusion may soon develop. The duration is variable. Death may occur in 1 to 2 weeks, or recovery be under way by this time; but in the cases which escape an early fatal termination, the convalescence is often much prolonged; and many patients are left with chronic lesions which may eventually prove fatal. Acute pericarditis may rapidly have dilatation occur as a complication, and cyanosis, edema, albuminuria, enlargement of the liver and hypostatic congestion of the lungs appear; or pneumonia may develop and be the immediate cause of death. In other cases already suffering from valvular heart-disease the occurrence of an acute pericarditis may fatally destroy the compensation.

The *prognosis* in children is in general unfavorable. Death may take place suddenly, or be preceded by evidences of failing compensation. Other cases recover from the acute symptoms but die later from sequels, depending upon pericardial adhesions. Even in rheumatic pericarditis, the most favorable form, Still[1] found only 1 case in 53 make a complete recovery, while 41 died within a few weeks or months. Not all statistics, however, present such an unfavorable outlook, and many of the milder cases of plastic pericarditis recover completely, at least so far as any immediate danger is concerned. The prognosis of the suppurative cases due to the pneumococcus or other germ is even more unfavorable. It is possible, however, for patients with purulent pericardial effusion to recover through operative interference. Cases in which pyopericardium is one of the symptoms of general sepsis end fatally.

Diagnosis.—Owing to the insidiousness of the onset and the presence of other diseases, the diagnosis of pericarditis is often difficult. The plastic form is recognized by the friction sounds; serous or purulent effusion by the alteration in the outline of the percussion dullness and the accompanying physical signs and symptoms. Radiography is a valuable aid in outlining the distention of the pericardium by fluid and the changes which take place in this from time to time, but dependence must not be placed on this alone. The differential diagnosis of fibrinous pericarditis is to be made especially from *plastic pleurisy*. It is possible for a dry pleurisy with inflammation of the external layer of the pericardium to produce a friction sound which suggests that of pericarditis. This, however, is not frequent, and the sound is more systolic than of the irregular type heard in pericarditis, and is influenced by the respiratory movement of the chest. Pericardial effusion of moderate degree may closely simulate cardiac *dilatation*. The peculiar shape of the dull area, with the presence of dullness in the 5th interspace to the right of the sternum, and the extension of the dullness to the left beyond the position of the apex-beat is characteristic of effusion. Change of position, too, produces in pericardial effusion a great alteration in the shape of the precordial dullness and of the intensity of the apex-beat on palpation, provided always that adhesions are not present which fasten the heart to the pericardial sac to a certain extent. The apex-beat is often displaced upward in effusion, and is moved downward and to the left in dilatation. A rapid increase in the precordial dullness points rather to dilatation than to effusion. The diagnosis from dilatation is often, however, very difficult, especially as both conditions may be present. A large exudate may simulate a *pleural effusion* of the left side. Careful study of the patient in different positions aids in the differentiation. The apex-beat of the heart is displaced to the right in pleurisy. The severity of the general symptoms in large pericardial effusion is much greater than in pleurisy. Yet in cases of large effusion the diagnosis is often very difficult, and I have seen the pericardial sac tapped from the axillary line under the impression that the condition was pleural effusion (Fig. 310). Purulent pericardial effusion is to be suspected chiefly from the

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[1] Common Disorders of Childhood, 1909, 448.

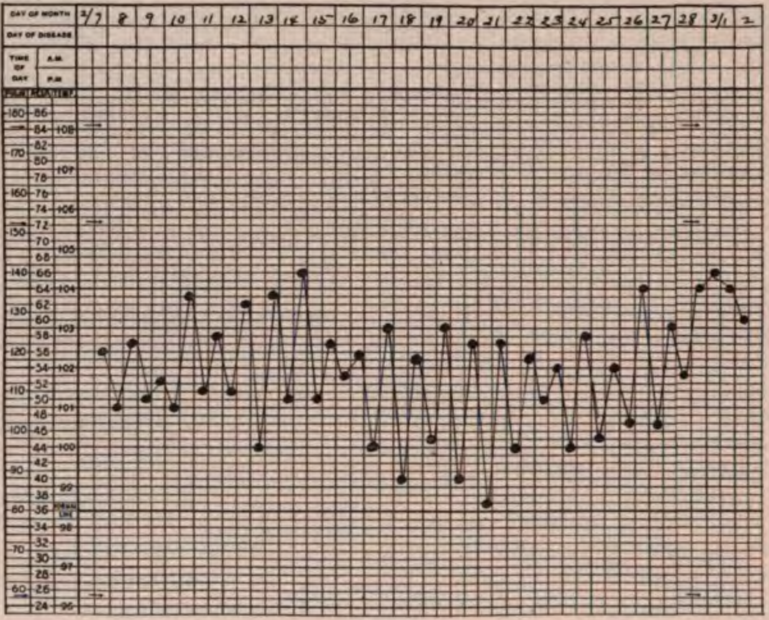

FIG. 310.—FATAL SERO-FIBRINOUS PERICARDITIS SIMULATING PLEURISY.

Delia B., aged 3 years. Illness began Jan. 20, with fever, dyspnea, and slight cough. Supposed to be pneumonia. General condition and cough grew worse. Entered Children's Hospital, Feb. 7, with rapid respiration, slight orthopnea, rapid, thready and irregular pulse, general condition very bad. Examination showed dulness on percussion on the left side anteriorly from the 3d interspace, downward, extending to the right nipple and well into the left axilla, but upper left axilla tympanitic; left paravertebral dulness; feeble respiration over the left side, heart-sounds distant, best heard at xyphoid, apex-beat not discoverable, condition believed at first to be pleural effusion. Tapping the 5th left interspace, anterior axillary line, gave slightly turbid, yellow fluid, result in the 4th interspace negative, tapping 5th right interspace close to sternum gave fluid, no germs found. Apex-beat returned after the aspiration; Feb. 13, aspiration of pericardium gave 16 ounces (473) blood-stained fluid, much relief; Feb. 24, signs of pneumonia in the right lung, pericardial fluid reaccumulating; Feb. 27, incision revealed pleural cavity empty, large amount straw-colored fluid, with slightly purplish tint, removed from the pericardium; Mar. 2, died.

character of the general symptoms, which suggest the presence of pus. Exploratory aspiration is, however, required to make a positive diagnosis from serous effusion. A small purulent pericardial effusion may sometimes be present without any physical signs although the symptoms are severe.

From *acute endocarditis* a plastic pericarditis is to be distinguished by the jerking, intermittent, and superficial character of the murmur, which does not keep exact time with the impulse. The murmur is also not conducted in any definite direction as in the case of endocarditis, and it is often influenced by change of position or by pressure with the stethoscope. At times, however, a pericardial friction is soft in character or is heard only during systole, and the diagnosis is then extremely difficult. In still other frequent cases there exists the combination of an endocardial and pericardial murmur. In such the diagnosis is best made by observing the differences in character over different portions of the precordium. Should pericardial effusion be combined with acute endocarditis the murmurs of the latter are combined with an enfeebling of the apex-beat out of proportion to the strength of the pulse. *Hydropericardium* resembles pericardial effusion in the signs elicited by percussion, but is readily distinguished by the accompanying conditions, such as hydrothorax or generalized edema.

Treatment.—This depends to a certain extent upon the nature of the cause and the variety of the disease. A patient with rheumatism should promptly receive anti-rheumatic treatment to prevent, if possible, involvement of the pericardium, and rest in bed should be insisted upon. If a plastic pericarditis has already developed the same treatment should be employed, although it is questionable whether it is of much benefit. The combined administration of bicarbonate of soda and the salicylates is recommended by many. Where the salicylates given internally derange the digestion, inunction with salicylate of methyl may be used. In all cases absolute rest in bed is imperative. The diet should be light and easily digestible if fever is present. Pain is to be relieved by the application of an ice-bag over the precordium and the administration of opium in sufficient dose. The same drug is useful in quieting cough. Evidence of cardiac weakness should be treated by suitable cardiac stimulants. Digitalis is objected to by some clinicians, and caffeine, strychnine and camphor are often to be preferred, sometimes best given hypodermically. For palpitation the ice-bag is often serviceable. If the accumulation of fluid in the pericardial sac is of moderate amount no treatment is required other than the symptomatic measures outlined. Blisters over the pericardium are to be avoided. In cases of large effusion the presence of threatening cyanosis and dyspnea may necessitate aspiration, but this should be deferred until actually imperative. Many of the cases of rapidly increasing pericardial dullness are in reality dependent upon dilatation, and the difficulty of reaching a positive diagnosis makes the chance of wounding the heart a very real one. When aspiration is to be performed, the needle is entered by preference in the 5th left intercostal space close to the sternum. The right interspace close to the sternum has been recommended, as has also the epigastrium, the latter especially by Marfan.[1] In cases where septic symptoms indicate the presence of a purulent effusion, exploratory aspiration is indicated for diagnostic purposes, but incision of the pericardial sac is the therapeutic procedure to be preferred. Indeed, for the relief even of serous effusion incision is a better operation than aspiration, and the latter may well be reserved for diagnosis only.

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[1] Bull. et mem. soc. méd. des hôp., 1911, XXXII, 133.

In the cases of pericarditis which recover every care must be taken to maintain the general and the cardiac strength, since dilatation is so likely to be present or pericardial adhesions to remain with their deleterious influence upon the heart. Continued rest in bed is necessary long after recovery from the inflammation has ceased completely. Even when the patient is out of bed precaution against over-exertion or undue excitement is to be used, with such symptomatic remedies as indications require.

CHRONIC PERICARDITIS

(Chronic Obliterative Pericarditis. Adherent Pericardium)

Etiology.—More or less adhesion between the opposite layers of the pericardium remains in probably every instance of acute rheumatic pericarditis. Indeed, these adhesions may sometimes be found at autopsy in the case of patients who had suffered from repeated attacks of rheumatism, with or without involvement of the endocardium, and without the existence of pericarditis ever having been recognized. Another form is that associated with a chronic mediastinitis. Adherent pericardium does not often follow purulent pericardial effusion, and the most common causes are rheumatism and tuberculosis, the former being very much the more frequent in early life. Pericardial adhesion was found by Lees and Poynton[1] in 113 of 150 fatal cases of rheumatic cardiac disease, and in 77 it was complete. It may occur at any age, but is much less often seen in infancy than later, and probably oftenest develops in later childhood, although perhaps not recognized until after this period.

Symptoms.—When there is but partial adhesion limited to a small area there may be no symptoms whatever at any time, and it is even possible for complete obliteration of the pericardial sac to develop unattended by clinical manifestations. As a rule, however, some evidences of the disease are present, and this is particularly true of the very frequent cases in which adhesions have also formed between the external pericardial layer and other tissues in the anterior mediastinum. The symptoms largely depend upon the interference with the development of the heart, and upon the myocardial changes which the thickened, adherent pericardium produces. Consequently those patients who showed no evidence of the disease after recovery from acute pericarditis may begin to do so months or even years later. Prominent among the symptoms is dilatation of the heart, nearly always present except in some of the tuberculous cases. The cardiac percussion-dullness is increased; with it is bulging of the precordium; and there frequently is a systolic retraction of the whole region, or limited to the apex-beat or the epigastrium. This is a very characteristic symptom when present, but is observed only in the cases where there exists also an adhesion of the external layer of the pericardium to the chest-wall, a condition developing oftenest when tuberculosis is the cause. The systolic retraction is followed by a visible diastolic expansion. There may be in these cases of *anterior mediastinitis* the presence of the pulsus paradoxus, and of diastolic collapse and inspiratory swelling of the jugular vein. The heart does not change its position by altering that of the patient. The pulse is rapid and later weak, and the apex-beat is often feeble. Cardiac systolic murmurs may be present. As the case advances the usual evidences of failing compensation develop, such as pallor, cyanosis, dyspnea, edema, and enlargement of the liver and spleen.

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[1] Med.-Chir., Transac., 1898, LXXXI, 445.

Prognosis.—The prognosis is always bad; death finally occurring as the result of increasing circulatory failure. The duration, however, is variable. Death may take place suddenly in cases where the disease was not suspected; or in others the fatal ending may come on suddenly or slowly after a few months or perhaps some years of continuance of symptoms.

Diagnosis.—When symptoms of adherent pericardium follow shortly upon an acute pericarditis the diagnosis is rendered easier; but in the majority a positive recognition of the disease is difficult or impossible, and the condition can be no more than suspected. The diagnosis rests upon the evidences of failing circulation and cardiac dilatation, combined with the systolic apical retraction should this chance to be present. The disorder is often confounded with chronic valvular disease, but may be distinguished by the absence of any characteristic endocardial murmurs. Some of the more chronic cases of pericardial adhesion show a resemblance in symptoms to cirrhosis of the liver. **Treatment** must be symptomatic and is unsatisfactory.

ACUTE ENDOCARDITIS

Etiology.—This is a frequent disorder in early life. Weill[1] places it as forming 5 per cent. of all diseases at this period. Not all statistics, however, give as high figures as this. In this country the incidence would seem lower. In 16,120 sick children admitted to two hospitals in Philadelphia and one in Boston the incidence of cardiac disease was 1.4 per cent. (Norris);[2] and Dusch[3] found chronic valvular disease in but 0.17 per cent. of 52,281 collected cases of sick children in various German hospitals. Among the *predisposing causes* age occupies a position of importance. The inflammation may occur in fetal life and be thus one of the causes of congenital cardiac anomalies. It is uncommon in infancy. Steffen[4] observed an exceptionally large number; 5 under 1 year in 45 cases in children, and Sutiagin[5] recorded 11 in the 1st year in 108 cases under 14 years. Such numbers are, however, certainly unusual. The age-incidence in 150 fatal cases reported by Lees and Poynton[6] was: up to 3½ years 1, first 5½ years 18, 5½ to 10½ years 105, 10½ to 12 years 27. Certainly after the age of 5 years the disease is as frequent as at any later period of life. It occurs rather more often in females than in males. Endocarditis may develop in the course of any of the infectious fevers, especially scarlet fever; or be associated with pneumonia or septicemia; but by far the most common cause is rheumatism. Including chorea in this category we find 73 per cent. of Weill's[7] 258 cases due to rheumatism. As already pointed out in discussing rheumatism (Vol. I, pp. 622, 625), endocarditis may follow promptly upon some other manifestation of the basic disease, such as arthritis, chorea, torticollis and tonsillitis; or may itself be the first and perhaps the only evidence of it. In this sense we may speak of endocarditis as being in some cases a primary affection, and it is possible that it may occasionally occur as a primary disease independent of rheumatism. The younger the patient the more liable is rheumatic arthritis to be followed by endocarditis, either in the first or in later attacks. Church[1] in analyzing 693 cases of acute rheumatism found the endocardium involved in 80 per cent. of those under 10 years; 69.164 per cent. of those from 10 to 20 years, and only 21.39 per cent. of those from 40 to 50 years. Half of the 140 cases of chorea examined by Osler[2] showed evidences of organic heart-disease 2 years after the attack.

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[1] Grancher, Traité des mal. de l'enf., 1904, III, 808.

[2] Arch. of Ped., 1906, July.

[3] Gerhardt's Handb. d. Kinderkr., 1878, IV, 1, 362.

[4] Klinik d. Kinderkr., 1889, III. Ref., Lempp, Monatsschr. f. Kinder., 1907–8, VI, 78.

[5] Dissert. Zurich, 1904. Ref., Lempp.

[6] Med. Chir. Transac., 1898, LXXXI, 443.

[7] *Loc. cit.*, 809.

In the matter of the *direct influence of infectious germs* as a cause of endocarditis no positive decision can yet be reached. Although the disease seems to be without doubt an infectious process no one germ is invariably present, and very frequently no microörganisms whatever can be discovered in the endocardium or cultivated from the blood. Most frequently found are the pneumococcus and forms of staphylococcus and streptococcus, but various other organisms have been observed.

Pathological Anatomy.—The lesions developing in antenatal endocarditis are situated nearly always upon the right side of the heart and oftenest at the pulmonary orifice. In cases arising after birth the left side is oftener attacked, especially the mitral leaflets. In 250 cases of endocarditis studied by Still[3] 241 showed only mitral murmurs. The lesions of endocarditis do not differ materially in early life from those found in adults. There takes place an increase of connective tissue near the surface of the valve or extending more deeply, with the formation of new blood-vessels and the growth of a granulation tissue, producing small vegetations (*verrucose endocarditis*). These vegetations may be completely absorbed and recovery follow, or may be transformed into cicatricial fibrous tissue with more or less consequent hardening and shrinking of the valve-leaflets and the production of permanent valvular lesions. In other, but infrequent, cases the granulation tissue may form a large mass, or may break down and an ulcerative process rapidly follow (*ulcerative endocarditis; malignant endocarditis*), extending to the papillary muscles and even the muscular tissue of the cardiac wall. In either condition clotting of blood may take place upon the leaflets and minute portions of this or of the vegetations themselves may be detached and carried into the circulation, this producing emboli in various parts of the body. This is not of frequent occurrence in childhood. I have, however, seen it on several occasions. In the verrucose and cicatricial forms bacteria may be found in the tissues or may be absent; in the ulcerative form they are present often in large numbers. Pericarditis is associated with the endocardial inflammation more frequently than in adult life. Some degree of myocardial change is generally present, dilatation of the heart is usually found, and hypertrophy may also be seen in cases which have passed into the chronic stage.

Symptoms and Course.—Two forms of endocarditis may be recognized clinically: (*a*) Simple endocarditis and (*b*) Malignant endocarditis.

(*a*) SIMPLE ACUTE ENDOCARDITIS.—Apart from the murmurs and the **symptoms** characteristic of the various chronic valvular lesions respectively, the evidences of acute endocarditis are not characteristic. The onset is more or less acute, with fever and general indisposition; sometimes vomiting and cough; restlessness; precordial pain; dyspnea; and rapid and irregular action of the heart (Fig. 311). This last is one of the most suggestive conditions in doubtful cases when no murmur is as yet audible. In other cases there are no subjective symptoms whatever. In the latter class, or if the disease develops in the course of other febrile disorders, the recognition of endocarditis is difficult unless constant supervision of the condition of the heart is exercised. If these repeated examinations are made the gradual development of a murmur may be discovered. This is usually soft, and is oftenest situated in the mitral area and accompanied by accentuation of the pulmonary second sound. Sometimes increase in the percussion dullness of the heart can be demonstrated; in other cases no such change can be detected.

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[1] St. Bartholomew's Hosp. Reports, 1887, XXIII, 269.
[2] Pract. of Med., 1909, 1050.
[3] Common Disorders of Childhood, 1909, 432.

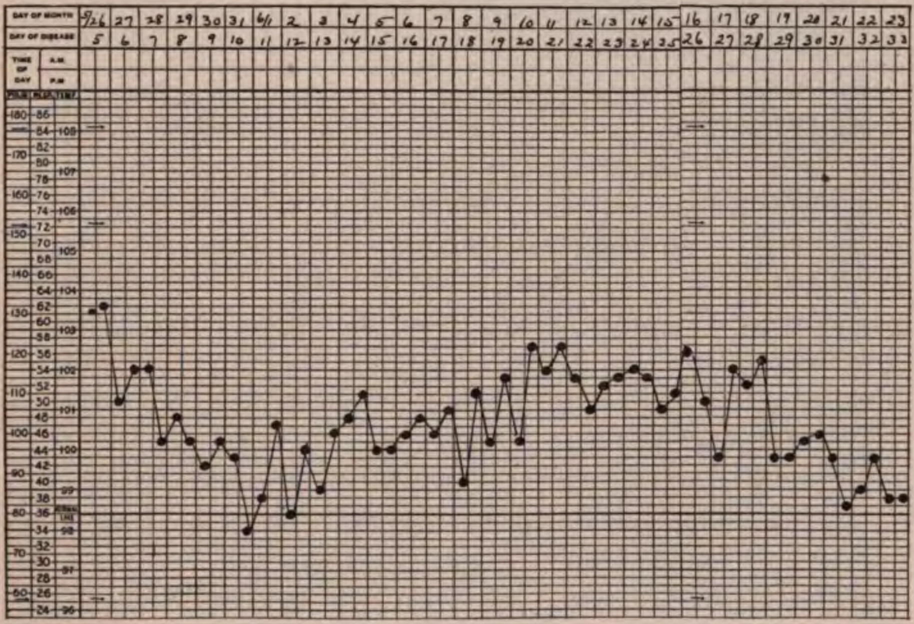

FIG. 311.—ACUTE MITRAL ENDOCARDITIS FOLLOWING OLD LESION, GRADUAL DEVELOPMENT OF AORTIC LESION.

Eva C., aged 11 years. Rheumatism 4 years previously. Been indisposed to active exercise. Taken ill 4 days before with vomiting, headache, fever, backache, marked dyspnea, precordial pain, and diarrhea. Entered Children's Ward, University Hospital, May 26 *Examination.*—Evidently seriously ill, nervous, restless, respiration 45–60, cardiac action rapid and irregular, loud, harsh systolic murmur over whole precordium, slight arthritis lower extremities, no edema; June 2, general condition gradually improved, but an aortic regurgitant murmur is developing; June 14, evidences of cardiac dilatation on percussion, dyspnea persisting, aortic regurgitant murmur harsh and louder; June 23, fever gone, slight dyspnea, double murmur present, removed to seashore.

The **duration** of the acute stage is from 2 to 4 weeks, by the end of which period in favorable cases the subjective symptoms have disappeared, and sometimes all traces of murmur as well and recovery is complete. This complete restoration to health is observed only in the mildest cases. The **prognosis** for entire recovery is not so favorable, and permanent valvular lesions remain, but usually no marked disturbance of compensation is seen until later. In occasional instances the lesion has been so great that failure of compensation immediately follows and persists, and death takes place in a short time from this cause. **Complications** are common and influence the prognosis unfavorably. Pericarditis occurs not infrequently (Fig. 312). Cardiac dilatation is present in all fatal instances, and some degree of myocarditis is very frequent in severe cases. Occasionally death is the result of embolic processes in the brain (see p. 340) or elsewhere, and not infrequently pneumonia is the cause of death. Usually, however, the prognosis of endocarditis is good so far as recovery from the acute attack is concerned; but it is not possible to determine early in the course how much permanent damage will remain. Of 148 cases of uncomplicated acute endocarditis of rheumatic origin reported by Dunn[1] 27 died during the attack, a mortality of 18.4 per cent. Even if recovery is complete there is still the very great probability that later attacks of endocarditis will occur, and that some of these will leave permanent impairment of compensation in their train.

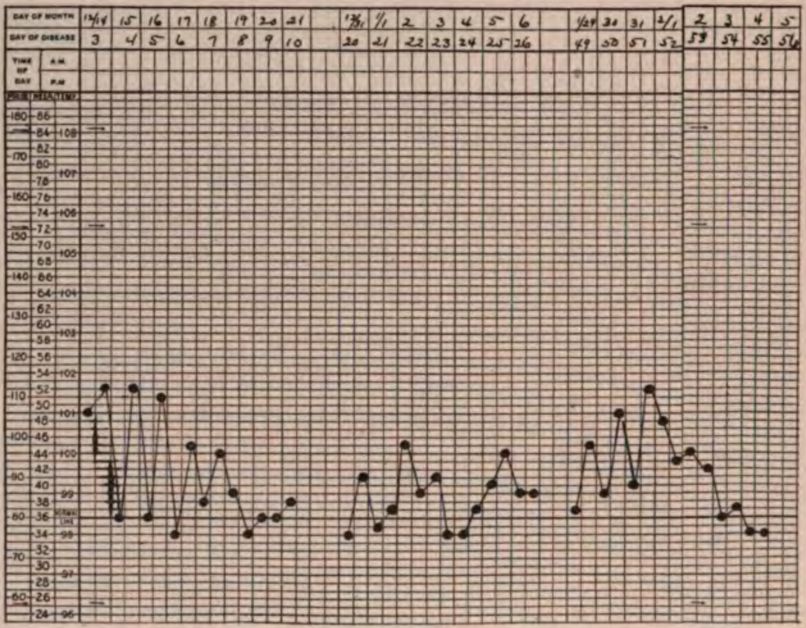

FIG. 312.—ACUTE ENDOCARDITIS FOLLOWED BY PERICARDITIS.

Joseph W., aged 9 years. Tonsillitis 10 days ago. Recovered in 4 days. Then, after exposure, developed pain in joints. 2 days ago severe precordial pain. Entered Children's Ward, University Hospital, Dec. 14, on 3d day of cardiac symptoms. Examination showed tumultuous cardiac impulse, physical signs of mitral insufficiency and stenosis, no arthritis, condition slowly improved, but murmurs persisted. Leaving bed Dec. 26 was followed in few days by increase of pulse-rate (120–130) and slight elevation of temperature. Sent back to bed. Jan. 29, rise of temperature followed by dyspnea, pain over heart, and distinct precordial friction; Feb. 3, general condition improved, friction almost inaudible, later slow failure of compensation and death followed May 3.

The **diagnosis** depends upon the evidence of disturbance of the cardiac action as described, combined with the physical signs of cardiac disease. Acute endocarditis is to be distinguished carefully from the *functional murmurs* which are so commonly heard in the course of febrile affections or at other times in children. (See p. 158.) Functional murmurs are

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[1] Amer. Jour. Dis. Child., 1913, VI, 109.

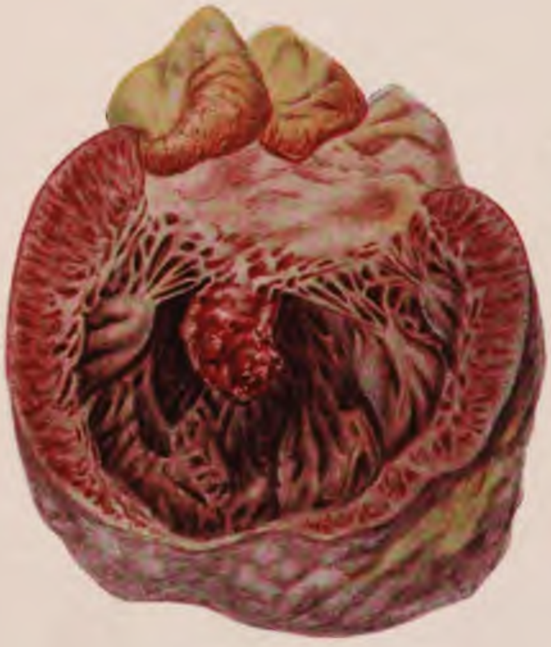

FIG. 313.—MALIGNANT ENDOCARDITIS.

Same case as in Fig. 314. Shows large mass of vegetations attached to the mitral valve.

usually soft, more distinct in the pulmonary area and at the base of the heart than at the mitral orifice, and are not transmitted as loudly to the axilla. There is also absence of undue accentuation of the pulmonary second sound and of cardiac hypertrophy. The diagnosis is often very difficult. It is to be noted that the functional murmurs which are most loud and diffuse are usually accompanied by decided anemia. Acute endocarditis is further to be distinguished from *pericarditis*. The subjective symptoms are very similar; but the murmur of endocarditis and the friction sound of pericarditis are generally different in quality, position, and rhythm. A fuller consideration has been given in discussing Pericarditis (p. 133).

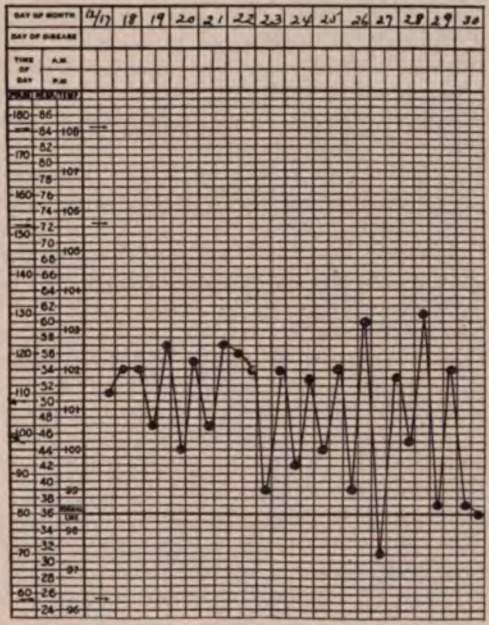

FIG. 314.—MALIGNANT ENDOCARDITIS.

Eileen K., aged 13 years. Gradual onset during 4 weeks, with malaise, drowsiness, fever, bed 4 days ago. Entered Children's Ward, University Hospital, Dec. 17. *Examination.*—Somewhat emaciated, general condition fair, scattered spots on body suggesting petechiæ, no edema, loud systolic murmur and thrill, moderate enlargement of cardiac area; Dec. 19, negative Widal, leucocytes 36,400, presystolic murmur also now audible; Dec. 29, general condition somewhat better, but abdominal pain and tenderness below liver; Dec. 30, became unconscious, developed right hemiplegia, pulse 160, weak and irregular, leucocytes 91,000, respiration moderately accelerated, lungs negative, liver enlarged, blood-culture gave streptococcus mitis. Death. *Autopsy* showed acute malignant mitral vegetative valvulitis, chronic aortic vegetative valvulitis, beginning bronchopneumonia and hemorrhagic nephritis with septic infarcts, degenerative changes in other regions. (See Fig. 313.)

(*b*) MALIGNANT ENDOCARDITIS.—This condition is distinguished pathologically from simple endocarditis only by the greater degree of bacterial involvement and the destructive process which follows. There is no sharp differentiation between the two varieties. Clinically, however, it is a quite distinct affection. The disease is rare in early life, only 47 cases having been collected from medical literature by Adams[1] occurring in subjects under 14 years of age. The heart of a very interesting case under my observation is shown in the illustration (Fig. 313). It may develop suddenly in the midst of health, occur in the course of some infectious malady, or be a complication of chronic valvular disorder. Sometimes it is seen in rheumatic cases instead of the ordinary simple form. The **symptoms** and physical signs are to some extent similar to those seen in acute simple endocarditis, but in general are of an indefinite, variable and puzzling nature. In some instances they are distinctly septic; in others typhoidal. The temperature may be continuously high or very irregular and intermittent (Fig. 314) and accompanied by sweating, delirium and the general evidences of sepsis. Septic emboli may occur in various regions with the varying symptoms characteristic of the locality, such as paralysis in different parts of the body, hematuria, intestinal hemorrhage, petechiæ, and enlargement of the spleen. In other cases diarrhea, tympanites, fever, petechiæ, sweating, and the development of the typhoid state may mark the typhoidal form. Sometimes the rapid production of cardiac dilatation and insufficiency, with murmurs heard oftenest over the mitral area but sometimes widely diffused, may aid in forming a diagnosis. In cases where the lesion is on the walls of the heart and not at the valve, no murmurs may be audible. The **duration** of the disease is variable, the range being from a few days to several weeks. The **prognosis** is very unfavorable, although there were 3 reported recoveries in Adam's 47 cases. The **diagnosis** rests upon the combination of the grave symptoms described with subjective and objective evidences of cardiac disease. The condition is to be distinguished from typhoid fever by the sudden onset, presence of leucocytosis, absence of the Widal reaction, irregularity of the fever, and possibly by the discovery of pyogenic germs in the blood.

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[1] Transac. Amer. Ped. Soc., 1902, XIV, 100.

Treatment of Acute Endocarditis.—As with pericarditis every precaution should be taken, as far as possible, to prevent the occurrence of endocarditis in patients suffering with rheumatism, especially insisting upon rest in bed. The chief element of treatment in a developed case is likewise absolute rest in bed in the recumbent position while the acute symptoms are present, in order to prevent the production of dilatation, or to relieve it if present. All excitement and exertion of any sort should be avoided. Even after acute symptoms are over rest in bed should continue for several weeks, as long as there is any reason to believe dilatation from myocardial changes exists or is liable to occur. The diet should be light and easily digestible. An ice-bag should be kept constantly over the precordium. This is useful in quieting the action of the heart and in relieving precordial pain and distress. Hydrotherapeutic measures may be of service to control fever.

As regards internal medication, it is a question whether any direct benefit is obtained by the use of the salicylates, but they may be tried if the digestion tolerates them. Pain may be relieved, when severe, by opiates; and restlessness by bromides. Digitalis or other cardiac tonics are needed only in cases where there seems danger of compensation being lost. This is usually not until late in the disease. The dose should be sufficiently large, but carefully watched.

The treatment of malignant endocarditis is symptomatic, like that of the simple form, but is usually valueless. The necessity of supporting the strength is great. Vaccine treatment with cultures of germs obtained from the blood may be tried.

CHRONIC VALVULAR DISEASE OF THE HEART

Etiology.—The sequel of acute endocarditis is chronic valvular disease, which follows in the large majority of cases, either after the first, or after some later attack. Sometimes the primary attack has been mild and overlooked and the first symptoms noticed are those of chronic valvular affection. Some of the causes operative in adult life, such as arteriosclerosis, diabetes, etc., are not etiologic factors in childhood. Tuberculosis, or rarely syphilis, occasionally gives rise to a slowly sclerosing process.

Pathological Anatomy.—The lesions are similar to those seen in adult life and consist of thickening, hardening, and shrinking of the leaflets, sometimes with adhesions connecting them. Calcareous salts may be later deposited in the valves. The change involves also the papillary muscles and the chordæ tendiniæ. The result of these processes is the production either of an inability of the leaflets to close the orifice (insufficiency) or of a narrowing of the orifice (stenosis), or of both. Consequent upon these conditions develop hypertrophy of the cardiac muscles in different regions, and later myocardial change and dilatation. It is noteworthy, however, that compensatory hypertrophy occurs more readily in early life than after this period, and that dilatation with failing compensation in chronic valvular disease, although common, is oftener deferred until later childhood or adolescence.

The valve affected in childhood is the mitral in the great majority of cases, although in a large proportion more than one valvular lesion is present. In 150 fatal cases reported by Lees and Poynton[1] the mitral valve was found involved in 98; the mitral, aortic and tricuspid in 32; the mitral and aortic in 15; all the valves in 4; and no lesion was detected in 1. It will thus be seen that there was involvement of the mitral valve in 149 of the 150 cases. In 262 cases of valvular disease dependent upon rheumatism, examined clinically by Dunn[2] some years after the first development of the lesion, there was found mitral insufficiency alone in 165; mitral stenosis alone 5, aortic insufficiency alone 1, mitral insufficiency and stenosis 79, mitral and aortic insufficiency 8, mitral insufficiency and stenosis with aortic insufficiency 4. Similarly in 676 cases of cardiac murmurs, apparently organic, studied by Priestley[3] there were, mitral regurgitant 562, mitral stenotic 27, mitral regurgitant and stenotic 49, tricuspid 5, pulmonary 26, aortic 7.

Symptoms.—The symptoms of valvular disease vary to some extent with the lesions, but depend for the most part on whether or not compensation is only slightly or not at all disturbed, or whether it is lost. In the former event children with valvular disease may long show no general symptoms whatever, and be about and indulge in active exercise without distress. In other cases with nearly complete compensation certain manifestations are noticeable; among them being moderate shortness of breath on exertion; sometimes palpitation; cough; epistaxis; indigestion, and headache. Very commonly there is an anemia which is very resistant to treatment, and a disposition of the patient to indulge in quiet games rather than active ones. This last is often very characteristic. The children make no complaint, not realizing that their inactivity is abnormal; and it may be only by accident or induced by the observation of these symptoms that the physician makes a physical examination and discovers the valvular disease. The duration of this period of compensation is very variable. Sometimes it is almost continuous for years until puberty is approached, when the increased rapidity of bodily growth may occasion its loss. Sometimes it is more or less intermittent; some temporary impairment of health from other cause disturbing the compensated cardiac condition, or the loss of compensation resulting from unusual physical exertion. Prominent among the causes of loss are recurring attacks of acute endocarditis. When failure of compensation does occur, the evidences of it are for the most part the same, no matter what the nature of the valvular lesion may be. There develop the general symptoms of venous stasis. There is cough; orthopnea; sometimes hemoptysis; epistaxis; edema (Fig. 315) first in the feet and then widespread and involving the serous cavities; enlargement of the spleen and liver; cyanosis; distention of the superficial veins; disturbances of digestion, and albuminuria with diminished secretion of urine. The heart is dilated; the apex-beat diminished in strength; and the radial pulse small. All these symptoms are the result of the stasis which the loss of compensation produces, but not all of them need be present at any one time, and they vary slightly with the lesion which exists; and all may disappear if compensation is restored.

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[1] Med.-Chir. Transac., 1898, LXXXI, 449.

[2] Amer. Jour. Dis. Child., 1913, VI, 104.

[3] Brit. Journ. Child. Dis., 1916, XIII, 353.

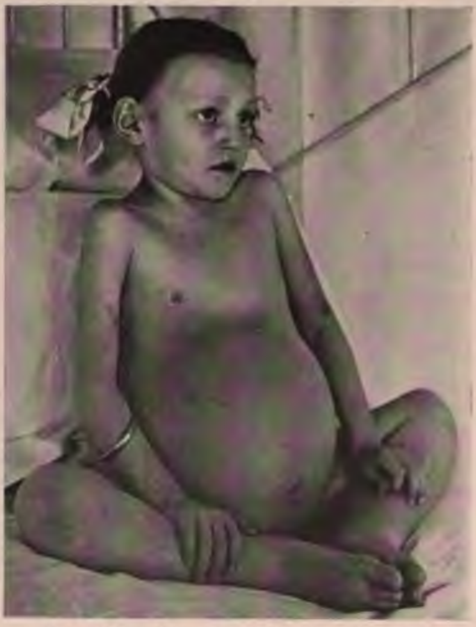

FIG. 315.—EDEMA OF CARDIAC DROPSY.

Child of 6 years in the Children's Hospital of Philadelphia. Ill 3 years with cardiac disease. Much improved later under treatment.

Clinical Forms and Physical Signs.—The physical signs vary with the valvular orifice affected and do not differ materially from those found in adult life. The different varieties of the disease and the symptoms exhibited may receive brief consideration in this connection.

Mitral Insufficiency.—This is by far the most frequent of the chronic cardiac lesions. It was found clinically in 241 of Still's[1] 250 cases, and in 124 of these there was a diastolic murmur as well, indicating a complicating mitral stenosis. The principal physical signs are a systolic murmur with maximum intensity oftenest at the apex, but widely transmitted and heard well in the axilla and at the back; accentuation of the pulmonary second sound; and hypertrophy, and later dilatation, of the right side of the heart and finally of the left as well. This latter is indicated by displacement of the apex-beat to the left, and downward as far as the 5th or 6th interspace. In children in whom the disease has lasted for some time there is often a very visible bulging of the precordium, and the cardiac dullness may be much broadened, reaching from the right of the sternum to the left anterior axillary line. Until decided dilatation has occurred there is a forcible impulse of the whole precordium and especially at the apex. With the development of dilatation the impulse grows feebler, and the murmur less distinct and even absent. Among the special symptoms are chiefly those of passive congestion of the pulmonary and of the general circulation, the latter dependent upon the regurgitation of blood through the dilated tricuspid orifice.

Mitral Stenosis.—This occurring alone is not common in childhood. It is found oftenest in combination with mitral insufficiency in cases where this disease has been of rather long continuance. Characteristic of it is the diastolic murmur limited to the usual small area in the neighborhood of the nipple. Combined with this are accentuation of the pulmonary second sound, enlargement of the right side of the heart, and diastolic thrill readily felt by palpation. The murmur is usually rough and ends abruptly with the first sound of the heart, or ceases before this occurs. The left side of the heart does not hypertrophy in uncomplicated cases, and in these the stenosis is not productive of many symptoms, the most characteristic being evidences of pulmonary congestion; such as cough and attacks of bronchitis, dyspnea, hemoptysis, and sometimes precordial pain, rapid pulse, and palpitation. General edema does not occur unless the tricuspid orifice dilates.

Aortic Insufficiency.—As stated this is a much less common affection in childhood, found chiefly in cases approaching puberty. This certainly, at least, applies to cases in which it can be recognized clinically. As a post-mortem finding the lesion is probably more frequent, since Lees and Poynton[2] reported 51 instances of involvement of the aortic valve, usually slight, in their 150 fatal cases of cardiac disease. Very exceptionally it is the only lesion present. The physical signs consist of a loud, widely diffused diastolic murmur with maximum intensity near the 2d right costal cartilage, and transmitted down the sternum or toward the apex. There is absent or enfeebled aortic second sound, decided hypertrophy of the left ventricle, often bulging of the precordium, displacement of the apex-beat downward, a strong but quickly receding radial pulse (Corrigan's pulse; trip-hammer pulse), and throbbing in the carotids. A capillary pulse may often be found in the finger-nails or the lips. No evidence of enlargement of the right chambers is present, unless mitral disease is a complication; or until, through failing compensation, general cardiac dilatation occurs, when a dynamic insufficiency of the mitral and tricuspid valves may develop. Cases in which aortic regurgitation is the only or the predominating lesion exhibit certain characteristic symptoms, among these being headache, dizziness, syncope, flashes of light before the eyes, precordial pain, anemia, epistaxis, and sometimes hemoptysis.

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[1] Common Disorders and Diseases of Childhood, 1899, 432.

[2] *Loc. cit.*, 448.

Aortic Stenosis.—This condition is even more uncommon in children than the last. Poynton[1] encountered it in only 2 of 500 cases of heart-disease in children. Consequently a systolic murmur heard at the aortic orifice must be determined only with great caution to be evidence of stenosis. The murmur is heard loudest at the aortic cartilage, and is transmitted into the vessels of the neck. The aortic second sound is weak or is replaced entirely by the diastolic murmur of an accompanying aortic regurgitation. The left ventricle is hypertrophied and later dilated as in aortic insufficiency. The symptoms are few, unless compensation is lost, when dyspnea, syncope and precordial pain are liable to develop.

Tricuspid Insufficiency.—This is practically a condition always secondary in post-natal endocarditis; there being no disease, but only a dilatation of the tricuspid orifice. It is seen most often accompanying mitral insufficiency, but is capable of developing in any disorder of the heart which produces dilatation of the right ventricle, among these being interstitial pneumonia, asthma, and emphysema. There is a systolic murmur heard loudest over the lower part of the sternum, and the jugular veins are dilated and sometimes show pulsation. The lesions following passive congestion are found in the liver, spleen and kidneys, and ascites is present.

Tricuspid Stenosis.—This is uncommon, and nearly always only secondary. In the analysis of 128 cases by Ashton and Stewart[2] there were none less than 11 years of age. It can scarcely be distinguished by physical signs and symptoms from mitral stenosis. There is dyspnea, but without evidence of pulmonary congestion unless from an accompanying mitral disease.

Course and Prognosis of Chronic Valvular Disease.—Under this heading may be considered both the prognosis in general and that of the special varieties of chronic valvular disease. As already stated a large number of cases developing in early life exhibit for a long time no functional disturbance; or only at such times as some accidental cause produces temporary loss of compensation. The occurrence of any severe acute disorder is liable to bring about a more serious disturbance of compensation and to effect the prognosis unfavorably, and acute dilatation takes place readily in early life. So, too, any condition producing a chronic impairment of the general health, such as insufficient food, unsuitable hygiene, and the like, is liable to exercise a harmful influence. Especially to be mentioned here is the absence of proper regulation of the life with regard to the amount of exercise which should be taken. Under some of these influences, or from the development of recurrences of acute endocarditis, to which children are especially predisposed, or as the result of complicating pericarditis or pericardial adhesion (see p. 140) there is a tendency to a gradual increase in the severity of the condition; and toward puberty, owing to the rapid growth of the body and the increased strain which this puts upon the heart, decided disturbance of compensation becomes more common and persistent. On the other hand, there is a remarkable ability in early life for compensation to be regained. Not many deaths occur until well on in later childhood; but after this the course is likely to be unfavorable in those children who have severe cardiac lesions, and not many of them reach adult life. When the lesion is but slight and well compensated and the general health is good, the chances are better. If such cases reach puberty with few if any signs of loss of compensation, the prognosis is not unfavorable; and it must not be forgotten that complete and final recovery with disappearance of all symptoms and physical signs may exceptionally take place, even in those children who have exhibited earlier what appeared to be positive evidences of chronic valvular disease lasting through several months or years.

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[1] Garrod, Batten and Thursfield Dis. of Child., 1913, 447.

[2] Amer. Jour. Med. Sci., 1895, CIX, 177.

The prognosis during the period in which symptoms are manifest depends more upon the state of hypertrophy or of dilatation of the heart respectively, the condition of the general health, and the severity and duration of the cardiac symptoms than upon the variety of lesion present. On the other hand, the prognosis in general, as to the duration of life and the symptoms which may develop, is influenced to some extent by the nature and extent of the lesion. Uncomplicated *mitral insufficiency* is readily compensated in childhood but less satisfactorily later. As compensation is lost and dilatation of the right ventricle occurs, tricuspid insufficiency develops with resulting venous stasis, first in the pulmonary circulation with passive congestion of the lungs and then in the general venous system. On the other hand, in cases of moderate insufficiency, which escape later attacks of endocarditis or pericarditis, a permanent compensation is not infrequently attained. *Mitral stenosis*, if uncomplicated, is of slow course and long in producing evidences of distrubed compensation. There is a tendency for the obstruction to increase as time passes, but life is often much prolonged. When, as is so frequently the case, it is associated with mitral insufficiency, the prognosis becomes that of the latter lesion. *Aortic insufficiency*, if alone or the predominating lesion, is capable of continuing for a long time with few symptoms and patients may reach a useful adult life. It is possible, however, for it to produce sudden death in later years. It may finally be attended by dilatation of the left ventricle and failure of compensation; oftener from the frequently complicating mitral insufficiency. On the whole its prognosis is unfavorable. *Aortic stenosis* offers often a favorable prognosis for the duration of life. Eventually dilatation of the left ventricle is liable to occur with mitral leakage and loss of compensation; but it is probable that in many of the cases of apparently secondary mitral disease, the mitral valve was attacked at the same time as the aortic.

The general mortality of chronic cardiac disease of rheumatic origin, as reported by Dunn,[1] and as based on the later history of 209 cases, is at least 23 per cent. and probably nearer 50 per cent.

Death in chronic valvular disease may sometimes be the result of cerebral or pulmonary embolism, or of sudden cardiac paralysis. Oftener it results from the occurrence of some intercurrent disorder especially of the pulmonary apparatus or the kidneys. Still oftener, and usually, it is brought about by gradual loss of compensation due to myocardial changes and cardiac dilatation.

Diagnosis.—On account of the slight development or entire absence of symptoms which is so frequently observed in children with chronic valvular disease, the diagnosis is often only to be made by a systematic examination of the heart in every child exhibiting manifestations of illness of any sort. A lesion having been discovered, the nature of this is to be decided. This depends upon the character of the physical signs found, and has already been considered. The general cardiac symptoms exhibited are an aid to a much less degree. The differentiation from congenital cardiac disease has already been discussed (p. 127). The distinguishing between organic and accidental murmurs often presents the greatest difficulty unless positive evidences of loss of compensation are present. (See Accidental Murmurs, p. 158.) This is especially true if decided anemia exists. It is important to avoid a hasty conclusion of the existence of valvular disease based upon murmurs alone, particularly if systolic, and even the evidence of cardiac enlargement may be inconclusive. Repeated observations extending over a considerable time may be needed. Never to be forgotten, too, is the normal accentuation of the pulmonary second sound which is characteristic of childhood. It is impossible to base upon this the diagnosis of increased congestion of the pulmonary circulation due to valvular disease, unless the accentuation passes the limit which may be called physiological.

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[1] *Loc. cit.*

Treatment.—This may be divided into: (1) that for failing compensation present; (2) that of a preventive nature to be employed in the effort to maintain compensation.

1. The **treatment for decompensation** is much the same as in adult life. The degree of its presence is a much more valuable guide than is the nature of the lesion. Absolute rest in bed is the first consideration and often alone will restore the compensation. Frequently it is not possible for the child to maintain a recumbent position on account of orthopnea, and propping in bed by pillows or the use of a bed-rest is necessary. Except in cases of very moderate severity the administration of some one of the cardiac tonics is indicated, if feeble pulse, diminished secretion of urine, and evidences of cardiac dilatation are present. Most reliable among these is digitalis, the tincture being my own preference. The dose given should be proportionate to the severity of the case, although children usually bear it in relatively large amount. An average dose would be 5 minims (0.31) given 3 to 6 times a day at the age of 10 or 12 years. It often deranges the gastric digestion, and in every case its action upon the circulation should be carefully watched. At the first sign of cardiac irregularity developing during its employment its administration should be abandoned for a time. Substitutes for digitalis may be given, one of the best being tincture of strophanthus in equal dosage. Convallaria is recommended by some. Caffeine and camphor are of service especially hypodermically when a speedy effect is desired, since that of digitalis is only slowly established (Worth Hale).[1] In cases with moderate loss of compensation the employment of the Nauheim treatment, with the carbonic acid baths and Swedish movements, is sometimes of decided service, and can readily be carried out in the hospital or the home.

Certain symptoms require special therapeutic measures:

Dyspnea is aided by the propping in bed referred to. In addition the origin of the dyspnea is to be sought for, whether dependent upon hydrothorax, pressure from a distended abdomen, or cardiac asthenia alone; and treatment for the cause instituted. Dropsy is often relieved by saline purgatives and diuretics. Among the latter the caffeine preparations such as diuretin and theocine are often useful, perhaps in combination with digitalis. When there is a large effusion into the pleural or peritoneal cavity aspiration of the fluid should not be too long delayed.

[1] U. S. Publ. Health and Marine Hosp. Service, Hygienic Lab. Bull., 1911, No. 74.

The heart will often respond to medication better afterward. Cyanosis is sometimes benefited by the combination of nitroglycerine with digitalis; although the arterial pressure is seldom high in children, and less aid is to be expected than in adults. Vomiting is often troublesome. In such cases the administration of digitalis by the mouth must be abandoned, and other cardiac stimulants employed. At the same time efforts must be made to relieve the vomiting by the administration of bismuth, lime-water, cracked ice, and the like; and, if necessary, morphine hypodermically. However, if the vomiting depends upon the passive congestion from cardiac weakness improvement of the strength of the heart is imperative. Tumultuous cardiac action is quieted by opiates better than by digitalis; and for diminished secretion of urine diuretic remedies and digitalis are required as for dropsy. Restlessness and sleeplessness may need bromides, veronal or opium. Precordial pain is aided by opium or nitroglycerine or the application of an ice-bag. As improvement begins rest in bed should continue, and cardiac tonics still be administered, although in diminished dosage. The leaving the bed and the resumption of excercise must be made very cautiously, using first a chair or a wheel chair. Then a little walking follows; but daily a considerable time should be spent at rest recumbent. Tonic remedies, especially iron, are often of value.

In cases where disturbances of compensation are frequent, or but of slight degree, some modification of the treatment described is needed, as continuous confinement to bed would not be advisable. The exercise must now be regulated with even greater care. Gentle, slow walking, graduated as in the method employed at certain health resorts; Swedish movements; and massage are serviceable, the choice depending upon the case. Carbonic acid baths, combined with passive resisted movements, according to the Nauheim method, are now of especial value. Change of climate is frequently beneficial and cardiac tonics, strychnine, cod-liver oil and often iron are to be employed.

2. The **maintenance of compensation** is by far the most important part of the treatment of cardiac disease, since it is much easier to compass than is the restoring of compensation which has been lost. The patient should always be under medical supervision, at least at intervals of a few months, even although the general health appears to be perfect. The whole method of life should be arranged in detail, yet refraining from creating the idea of invalidism, unless the natural carelessness of the patient or the degree of restraint needed actually compels this. The time should be spent largely in the open air; the hygiene of the dwelling carefully regulated with avoidance of confinement to close, dark, or illy ventilated rooms; the clothing should be warm in winter, preferably of wool, and in summer not of a nature to permit of sudden chilling or of overheating. The diet should be abundant and digestible with the avoidance of coffee, tea and cigarettes. Mental over-work and undue excitement must be shunned. The child should retire early and obtain an abundance of sleep. Not every child who exhibits physical signs of valvular disease must be forbidden the partaking in active games and sports. It is as imperative to keep the cardiac muscle in good condition by sufficient exercise as it is to avoid weakening it by over-exertion. It is particularly those exercises which throw long-continued, severe strain on the heart, or those involving sudden violent strain, which are to be avoided; but all other forms are to be encouraged in suitable subjects. Everything, in fact, depends upon the individual case, and only by a

tentative increase of exercise under careful supervision can the proper limit be determined.

Whenever the slightest indication of cardiac over-strain appears, a few days or a week in bed is advisable; and in very many instances a serviceable procedure is the systematic resting recumbent for an hour in the middle of the day. This is particularly true of cases showing any degree of general debility or anemia.

The possibility of the contraction of any of the acute infectious diseases is to be shunned as far as possible, since they are so likely to disturb the compensation; and the avoidance of rheumatism by the seeking of warm climates in winter and spring is equally important. Especially about the time of puberty, with the increase of physiological strain put upon the heart by the rapid growth, precautions must be taken against over-exertion, either bodily or mental. During adolescence the selection of a future occupation often becomes a matter of moment. When possible, one is to be chosen which combines fresh air with moderate exercise without undue exertion.

As regards medication no cardiac tonics are required in cases of fully established compensation. The practice of giving such drugs simply because a murmur is heard is to be condemned. Great care, however, must be taken to watch for and treat promptly the anemia which is so common in chronic valvular disease. Iron alone or combined with arsenic is serviceable here, and is sometimes more efficacious when given hypodermically. (See Anemia, p. 460.)

MYOCARDITIS

Etiology.—As a secondary disorder this is not infrequent in early life, although much less often seen than in adults. As a primary affection it is very rare. Prominent among the causes are the acute infectious diseases, especially diphtheria, scarlet fever, and typhoid fever, through the action of the toxins produced by them on the muscles of the heart. Acute endocarditis and pericarditis generally have some degree of myocarditis attending; while chronic valvular disease, and especially chronic adhesive pericarditis, may have it as a final and fatal complication. The frequency with which myocardial alterations attend acute rheumatic inflammation of the heart is probably very great. Exceptionally a form of myocarditis occurs in septic conditions of any sort, and depends upon the entrance of pyogenic bacteria of different kinds into the cardiac muscle and the development of abscess-formation there. Syphilitic and tuberculous myocarditis are also encountered.

Pathological Anatomy.—The disease may appear in one of two forms, or in a combination of them—the *parenchymatous* and the *interstitial*. The first, usually acute, is the milder, and is that commonly produced by the infectious fevers. The muscle-fibres lose their transverse striation and are filled with albuminous granules; while a hyaline transformation may be seen is some places. Often the granules are to a large extent fatty in nature, especially later in the disease. Sometimes there is complete breaking up of the muscle-fibres. The process is circumscribed or more frequently diffuse. The lesions are degenerative rather than inflammatory. Macroscopically the heart is soft, flabby, friable, pale, and not uncommonly dilated. The muscle is grey in color, often with yellow and hemorrhagic streaks.

The acute interstitial form is of much rarer occurrence. It may accompany the lesions described, or result through extension from a pericarditis or endocarditis, or depend upon the entrance of bacteria through the blood-vessels of the heart. The process is usually localized in numerous small areas. The connective tissue between the muscle-fibres and about the small blood-vessels exhibits a small-celled infiltration, which may in septic cases advance to the formation of minute abscesses in the wall of the heart. In chronic interstitial myocarditis whitish or yellowish opaque indurated areas are produced in the cardiac wall, and these may later give rise to cardiac aneurism and even rupture. Syphilitic processes in the heart belong to this form of interstitial myocarditis.

The condition much most frequently found is a parenchymatous myocarditis combined with a certain amount of interstitial alteration; the muscles, connective tissue, nerves, and even the blood-vessels being involved in the inflammatory and degenerative changes.

Symptoms.—The clinical features of myocarditis are those of cardiac weakness. Whether or not this weakness depends upon myocarditis it is often impossible to determine before autopsy is made. The symptoms consist of rapid and weak or sometimes unusually slow pulse, palpitation, pallor, dyspnea, feeble valve-like cardiac sounds, syncope, disturbance of digestion, and evidences of dilatation such as enlargement of the heart and the development of murmurs from the increased aperture and incomplete closure of the orifices. Evidences of venous stasis may finally appear. In many instances myocarditis may be found at autopsy in cases in which there were no clinical signs of it during life.

Course and Prognosis.—The prognosis is always grave, but dependent upon the cause. After the infectious fevers death may take place suddenly or occur in a very short time without any distinct cardiac manifestations; but, on the other hand, complete recovery may follow slowly even after very threatening symptoms. In other conditions, as in the presence of pericarditis or endocarditis, the symptoms may develop slowly and advance to a fatal ending. In the chronic cases the prognosis is most unfavorable.

Diagnosis.—When symptoms of cardiac failure come on rapidly in the course of an infectious disease, especially diphtheria, without the evidence existing of a pericarditis or endocarditis, it is probable that myocarditis is the cause. Yet a diagnosis which is beyond doubt is seldom possible during life.

Treatment.—Most important is that of a prophylactic nature. Caution should be used in cases of diphtheria, or of any long-continued severe case of other infectious fevers, that the patient's strength is well-maintained and a recumbent position rigorously observed. If symptoms develop, the necessity for absolute recumbency, freedom from all excitement, and the employment of sustaining remedies is even greater. Strychnine is sometimes of service, and digitalis to a less extent; but the latter should be used cautiously and in small doses. If urgent symptoms are present the hypodermic administration of caffeine, camphor or often morphine may be useful. Alcoholic stimulants may be given freely during the emergency.

DILATATION OF THE HEART

Enlargement of the heart by increase of the thickness of its walls (*hypertrophy*) is occasionally a congenital condition, but commonly results as a compensatory process, and is in that sense salutory and not to be looked upon as a disease. Enlargement of the cavities of the heart, however, (*dilatation*) is always to be considered a pathological condition.

It may occur with or without thickening of the cardiac walls. The boundaries of the normal heart have already been considered (Vol. I, p. 55).

Etiology.—Among the frequent causes is myocarditis, produced by toxic or other influences arising in the course of acute rheumatic inflammation of the heart or of acute infectious fevers; but it results also from pericardial adhesion, and chronic valvular disease is uniformly attended by more or less dilatation in cases with failing compensation. Nephritis and, very rarely in childhood, arteriosclerosis lead to hypertrophy which may eventually have dilatation follow. Anemia, pertussis, general malnutrition, and chronic pulmonary diseases, especially chronic bronchopneumonia, are often attended by a moderate degree of dilatation. Overexertion is not a frequent cause in early life.

Symptoms.—The condition may be acute or chronic; the former being seen especially in rheumatic carditis and in infectious diseases, and being sometimes sudden in onset; the latter consequent upon chronic valvular disease and pericarditis. The symptoms consist of dyspnea; feeble, rapid pulse; diminished arterial tension; diffuse, fluttering apex-beat; cyanosis; pallor; cough; palpitation; edema, and effusion into the serous cavities—in fact, the usual symptoms of failing cardiac compensation, since dilatation is the accompaniment of this condition.

The **physical signs** on percussion vary with the cause. Usually both sides of the heart are involved, although the right side is generally the first to suffer. In dilatation of the right chambers the percussion boundary is displaced to the right. In left-sided dilatation the heart extends too far to the left and downward. Auscultation reveals a weakened, short first sound and an accentuation of the second sound, and a soft systolic murmur is not uncommon. This is of dynamic origin and depends upon the incomplete closure of the orifices, the result of the widening of these which has taken place.

Prognosis.—This depends upon the cause. Acute dilatation is always dangerous. Chronic dilatation is often removable if sufficient compensatory hypertrophy can be established. When moderate and dependent upon conditions other than pericarditis or valvular disease the prognosis is usually good.

Diagnosis.—The diagnosis of dilatation from pericardial effusion is often extremely difficult, especially as the conditions may be combined. In effusion the percussion-outline of the heart is of a peculiar shape, dullness existing in the 5th interspace to the right of the sternum; while in dilatation this is usually not the case. The apex-beat is displaced upward in effusion, to the left and downward in dilatation; and in the former the force of the radial pulse is out of proportion to that of the apical impulse.

Treatment.—When dilatation is dependent upon chronic valvular disease, the treatment for this latter is indicated. The moderate dilatation which results from anemia or similar conditions is to be treated by remedies directed to the primary disease. Acute dilatation requires the management indicated for urgent symptoms in acute myocarditis.

CHAPTER IV

FUNCTIONAL CARDIAC DISORDERS, MORBID GROWTHS, AND DISEASES OF THE BLOOD-VESSELS

FUNCTIONAL DISEASES

Here are to be included disturbances of function of various sorts, not directly dependent upon any known anatomical alteration of the cardiac muscle or valves, except the association at times of some temporary dilatation.

Etiology.—The causes are varied and not always clear. The tendency to the condition increases as the subject enters the period of later childhood. Among the acute causes are anemia; the existence of a febrile affection; general malnutrition; indigestion; various neuroses; an irritable heart-muscle; over-work at school; fright or other psychic disturbance; and the use of tobacco, tea or coffee.

Symptoms.—There exist great differences in the nature of the functional disturbances. Some of the more important of them may be enumerated:

Tachycardia.—Many of the causes mentioned may be operative here. The tachycardia may be paroxysmal or more continuous. In the *paroxysmal* form, if severe, the attack is characterized by rapid action of the heart, oppression, pallor, weakness, sweating, dyspnea and even orthopnea, and perhaps a slight cyanosis, and the child looks ill. The pulse may reach a rate of 200 or over. The seizure may last several hours or but a few minutes and some degree of dilatation may develop. It may occur at any age, but the frequency in childhood is not very great. Perhaps the youngest case reported was that by Hutchinson and Parkinson[1] in a boy of 2¾ years. The more *continuous* form may last several days, the principal symptom being the rapid pulse without marked disturbances of other nature. The disease in either form comes on in repeated attacks, the whole extending sometimes over several years. A few cases are reported (Hochsinger)[2] in which tachycardia had been continuous without intermission for over a year, the cause being probably a compression of the vagus by enlarged bronchial glands.

Palpitation.—This is frequently confounded with tachycardia under the impression that palpitation indicates a rapid cardiac action. As a matter of fact, palpitation is the perception by the patient of an uncomfortable, disturbed cardiac action which is, it is true, oftenest rapid but which may be simply irregular. In addition to the consciousness of the disturbed rhythm, there is a feeling of oppression; sometimes shortness of breath, and perhaps orthopnea; and in some cases dizziness, paleness and profuse perspiration.

Bradycardia.—This is often seen during convalescence from some of the infectious fevers, particularly typhoid fever, and without there being reason to believe that it depends upon a myocarditis, although this latter disease is a frequent cause of bradycardia of organic origin. It is common after diphtheria, and then probably is due to myocarditis. It often occurs, too, during tuberculous meningitis and in jaundice. The pulse may fall to 50 or 60 per minute or less. The condition may last for a number of days. It is usually combined with arrhythmia.

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[1] Brit. Jour. Child. Dis., 1914, XI, 241.

[2] Pfaundler and Schlossmann, Handb. d. Kinderheilk., 1910, III, 489.

An interesting form of bradycardia is that of **Stokes-Adams disease or Heart-block.** Although this is usually not a strictly functional disturbance, but depends, at least in some cases, upon actual pathological changes of certain of the muscular fibres of the heart, the bundle of His, it may conveniently be referred to in this connection. The attack is paroxysmal in nature and presents the complex of slow pulse; a disturbed cerebral state, such as syncope or convulsions; and auricular contractions more rapid than those of the ventricle. The disease is one of adult life, but has been reported in childhood, as by Schuster[1] in a girl of 4 years; Starck,[2] boy of 5 years; Armstrong and Monckeberg,[3] boy of 5 years; Hecht,[4] child of 9 years; Zahorsky,[5] child of 15 months. Whipham[6] recorded an interesting instance of the condition in an infant of 18 months, confirmed by an electro-cardiogram, which he regards as of congenital origin.

Arrhythmia.—This in moderate degree is so common in early life that it may be regarded as physiological. Friberger[7] in 321 subjects in later childhood found no instance in which the pulse was entirely regular. It is especially well marked under the same conditions which produce bradycardia, and is also seen without this in chorea, gastrointestinal diseases, in children of a nervous disposition, and after the use of tobacco, digitalis or some other drugs. It may be more or less constant, or present only during sleep or when recumbent, disappearing when the child sits up or leaves the bed.

Syncope.—Fainting attacks are not of uncommon occurrence, although comparatively less frequent than in adult life. They are not often associated with any actual weakness of the heart; in fact children with organic cardiac disease do not often have syncope among the symptoms. The nature of the attack is frequently of nervous origin, and the condition is to be considered as a functional disorder. The attacks are most liable to appear in nervous, debilitated, anemic children, or those with digestive disturbances, or suffering from the strain of school-life. Girls are more frequently the subjects than boys. The attack does not differ from the condition as seen in adults, being ushered in by pallor, yawning, dizziness, and feeble pulse, and terminating in partial or, less often, complete loss of consciousness. It lasts a few minutes up to over a half hour. It is most likely to occur before breakfast, or before going to school (Hutchinson).[8]

The condition is only to be distinguished from some of the milder attacks of petit mal. In these, however, the duration is much shorter, and there is not the very evident weakness of the circulation. The treatment of the attack itself consists in placing the child in the recumbent position with the head low, and administering a stimulant if necessary; although the faintness will soon pass off of itself. As a preventive measure tonic remedies, change of air, and removal from school are indicated, together with the treatment of any direct cause which may be apparent.

Accidental Cardiac Murmurs.—These are frequently present in early life, may be quite loud in cases of marked anemia, and are often heard, too, in acute febrile conditions; but they are found also in many other instances where explanation is entirely lacking. Some of these are cardiopulmonary in nature, the result, according to Hochsinger[1] of the impulse of the heart against the border of the lung and the production of a systolic vesicular sound; but it is likely that the majority are certainly not occasioned in this way, and the method of causation is much discussed and seemingly varies. The thin character of the blood in anemia is probably one cause; relaxation of the papillary muscles and temporary widening of the mitral orifice another. Hamill and LeBoutillier[2] found accidental murmurs present in 66.2 per cent. of 80 children examined.

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[1] Deutsch. med. Wochenschr., 1896, XXII, 484.
[2] Monatsschr. f. Kinderh., 1903–4, II, 11.
[3] Deutsch Arch. f. klin. Med., 1911, CII, 144.
[4] Ergebn. d. inn. Med.u. Kinderh., 1913, X, 324.
[5] Intern. Med. Jour., 1915, XXII, 57.
[6] Brit. Jour. Child. Dis., 1915, XII, 321.
[7] Arch. f. Kinderh., 1912, LVIII, 30.
[8] Brit. Journ. Child. Dis., 1916, XIII, 163.

The larger portion of the murmurs heard before the age of 3 years are, according to Hochsinger, cardio-pulmonary, while those of endocardial origin are most frequent in later childhood. Lüthje[3] examined 854 school children and found a functional systolic murmur in 612 (71.6 per cent.). The accidental murmur is nearly always systolic in time, and is heard best over the base or at the left border of the heart or at the pulmonary cartilage. It is often most evident at the end of expiration and when the patient is in the recumbent position, disappearing during full inspiration. It is usually not loudly transmitted to the axilla and seldom to the back, but in severe cases of anemia it is widely diffused, and is audible over the carotid and subclavian arteries, and is then frequently accompanied by a venous hum heard at the base of the neck. There are either no symptoms at all, or only those of anemia, if this is the cause.

Prognosis.—The prognosis of functional disorders of the heart is usually good, since the symptoms do not, as a rule, depend upon any organic lesion of any part of the body. It may be years before the disturbance disappears, but it will eventually do so if the cause is removed. Often the establishment of puberty witnesses this recovery.

Diagnosis.—The diagnosis of functional disturbances of the heart from those dependent upon organic cardiac lesions presents often great difficulty, and only a careful study of the symptoms and possible causes can succeed in distinguishing them. It is especially perplexing in the case of accidental murmurs. The recognition of these murmurs depends chiefly on the absence of other signs of cardiac disease, especially undue accentuation of the pulmonary second sound and of enlargement of the heart. They are usually soft, low-pitched and blowing, and do not replace any of the cardiac sounds. Often only the continuous observation of the case can lead to a correct conclusion. The employment of the electrocardiograph will be of aid in distinguishing the functional murmurs from those depending upon myocarditis. The cardiopulmonary murmurs are supposed to be characterized by their cessation if respiration is momentarily stopped at the end of expiration; while accidental murmurs of endocardial origin are not affected in this way. It is questionable whether it is possible to make any such sharp differentiation.

Treatment.—The cause should be carefully sought for and the proper treatment for this instituted. During attacks of arrhythmia or tachycardia the patient should be at rest, and nervous sedatives, and, if necessary, opium administered. Attention should be paid to the digestion. An ice-bag over the heart is sometimes useful. Digitalis may aid in cases at all long-continued. On the other hand, in many cases of bradycardia and arrhythmia following febrile diseases, getting the patient out of bed is the best treatment. This appears to produce a certain degree of stimulation which the heart requires; but is, of course, not suitable to cases where the severity of the preceding disease makes the existence of myocarditis probable.

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[1] Arch. f. Kinderh., 1913, LX–LXI, 377.

[2] Amer. Jour. Med. Sci., 1907, CXXXIII, 55.

[3] Med. Klinik, 1906, II, 404.

MORBID GROWTHS OF THE HEART

These are of extremely uncommon occurrence. In 28 cases collected by Quain[1] only 2 were in children. The most frequent form is lymphosarcoma extending to the heart from a primary growth in the mediastinum. The symptoms produced are those of pericarditis with effusion, and the fluid withdrawn is commonly blood-stained. Syphiloma of the heart has also occurred in children, and echinococcus has been reported.

DISEASES OF THE BLOOD-VESSELS

None of these are common in early life, including even those of a congenital nature, such as the narrowing of the isthmus of the aorta (p. 124) and the general hypoplasia of the aorta and peripheral arteries described by Virchow.[2] (See pp. 124 and 461.) **Arteriosclerosis with atheroma** is rare, yet probably more common than ordinarily supposed. Although it has been found even in the fetus, the most frequent period in childhood is shortly before puberty. It may be associated with the very unusual cases of chronic interstitial nephritis occurring in childhood, is sometimes seen as a result of congenital syphilis, or may occasionally occur after rheumatism or infectious diseases. The subject has been exhaustively reviewed by Fremont-Smith,[3] and Saltykow.[4] **Acute arteritis** of the peripheral vessels may occur as the result of various infectious diseases, syphilis, and rheumatism, and produce narrowing or occlusion of the lumen. It may also follow the formation of a septic thrombus or embolus, and a secondary aneurism may result. An **aortitis** may very exceptionally be due to rheumatism, syphilis, or some infectious process (Marfan).[5] Levy-Franckel[6] collected 35 instances of chronic aortitis and atheroma, including some of his own, occurring up to the age of 17 years.

Aneurism of the aorta is of very rare occurrence in early life. LeBoutillier[7] made a careful review of the published cases, equalling, with 1 of his own, 16 not over 12 years of age, including 1 occurring in fetal life reported by Phänomenow.[8] The youngest, excluding this last, was in a child of 2 years. The causes are various, among them being syphilis, rheumatism, and trauma, but oftenest the origin is obscure. **Aneurism of the peripheral vessels** is also rare. Instances, however, of its occurrence in different regions are reported, principally in the vessels of the brain. It is dependent oftenest upon syphilis; sometimes on sepsis.

Embolism is uncommon in early life. It is sometimes seen in acute endocarditis as a result of small thrombi leaving the heart and being carried by the blood to the brain or other parts of the body. I recall 2 very typical instances of cerebral embolism attending acute endocarditis in children of 11 and 13 years respectively. **Thrombosis** of the arteries is uncommon. It may form in severe cases of infectious fevers, pneumonia, from atheroma, or in conditions of great debility. I have seen gangrene of the leg produced by thrombosis of the artery in diph-

[1] Ref. Bruce, Keating's Cyclop. of the Dis. of Child., 1889, II, 813.
[2] Über die Chlorose und Anämie der Gefässapparat, 1872.
[3] Amer. Jour. Med. Sci., 1903, CXXXV, 199.
[4] Correspondbl. f. Schweiz. Aerzte, 1915, XLV, 1057.
[5] Sem. méd., 1901, XXI, 97.
[6] Arch. d. mal. du coeur, des vaiss. et du sang, 1912, V, 625.
[7] Amer. Jour. Med. Sci., 1903, CXXV, 778.
[8] Arch. f. Gynäk., 1881, XVII, 133.

theria, and again in a case of exhaustion resulting from unusually severe recurrent vomiting. Large thrombi are not infrequently found in the heart at autopsy upon greatly debilitated subjects or those with infectious or cardiac diseases. They form usually just before or after death. Very rarely thrombosis of the aorta may develop at an earlier period. Thrombosis of the veins is more common. It may occur as a result of an infectious **phlebitis** or in anemia, pneumonia or conditions of great debility, or from pressure. Any of the large venous trunks of the body may be affected, oftenest those of the lower extremities. David[1] reported an instance of double phlebitis of the lower extremities in a new born, following umbilical infection. **Thrombosis** of the superior or inferior vena cava has been observed repeatedly, the cause being marantic conditions, mediastinitis, or the pressure of intra-abdominal growths or of enlarged tracheobronchial glands. The jugular veins are also sometimes affected. The most frequent seat is the venous sinuses of the brain. (See p. 341.)

Dilatation of the veins, not of the nature of varicosities, is of very common occurrence in early life. It is seen in rachitis or in other condition attended by anemia or debility. Situated in the veins of the scalp it is sometimes very marked in hereditary syphilis. Dilatation of the veins of the abdominal walls often attends abdominal tuberculosis or morbid growths. Dilatation of comparatively small groups of vessels is seen in nævi and the like. (See Diseases of the Skin.) An extensive and excessive congenital or developmental dilatation of the arteries and veins is a rare condition, associated with hypertrophy of the limb in which it is situated. The subject has been reviewed by Parkes Weber.[2]

Symptoms.—The symptoms of the various lesions of the blood-vessels vary with the situation and do not differ from those seen in adult life. Reference only may be made to the frequency of a venous hum heard in the jugulars or beneath the upper part of the sternum. Landis and Kaufman[3] found it present in 84 out of 90 children examined, and could discover no special relationship to anemia. In adults the murmur was much less frequently heard, and generally in connection with anemia. This confirms the observations of Sawyer[4] and of Coombs.[5] (See p. 119.)

RAYNAUD'S DISEASE

(Symmetrical Gangrene)

Etiology.—The disease described under this title is probably a vasomotor neurosis, and might well be classified either with nervous disorders or with diseases of the skin. It is not a common affection at any period, and is less frequent in childhood than later. In 168 cases analyzed by Casserer,[6] there were 22 under 5 years and 8 between 5 and 10 years, and the affection may even occur in early infancy. The causes are not clearly understood. A familial tendency is observed in some cases. Congenital syphilis has been claimed to be the causative agent, and in some instances the earlier occurrence of an acute infectious disorder. In other respects the previous state of the health appears to be without influence. The immediate cause of an attack often is exposure to cold, even of only moderate degree.

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[1] Archiv de méd. des enf., 1913, XVI, 615.

[2] Brit. Jour. Dis. Child., 1918, XV, 13.

[3] Arch. of Ped., 1912, XXIX, 88.

[4] Brit. Jour. Child. Dis., 1910, VII, 310.

[5] Brit. Jour. Child. Dis., 1910, VIII, 109.

[6] Die vaso-motorisch-trophischen Neurosen, 1901; Ref., Garrod, Batten and Thursfield Dis. of Child., 1913, 504.

Pathology.—As far as this is known there appears to be a spasm of the smaller arterioles accompanied or followed by dilatation of the smaller veins and of the capillaries. In some cases hyperplasia of the wall of the arterioles has been found, and in others evidences of endarteritis, while in still others a neuritis has been discovered. There are also the lesions of ulceration and of gangrenous destruction of the tissue seen in severe cases.

Symptoms.—The local symptoms are those produced by interference with the circulation. This is seen oftenest in the hands or in some of the fingers; less frequently in the feet, or the ears or nose. The lesions are generally symmetrical in distribution. The first stage is that of local syncope, in which the affected part becomes white and cold, and a sensation of numbness, prickling, or tingling develops. In the mildest cases this lasts a few minutes or hours, and is followed by redness and throbbing, and a sensation of pain. In the more frequent severer cases a condition of local asphyxia follows the syncope, or may occasionally occur without this. The region involved now becomes painful, cold, and very cyanotic. This lasts some hours and is followed by an acute congestion, the result of the relaxation of the spasm, accompanied by very severe pain. In the cases of still greater severity the process goes on to the production of superficial necrosis, or even, in unusual instances, of gangrene with loss of the affected part. Sometimes instead of the involvement of the terminal portions of the extremities, or in addition to this, patches develop on other parts of the limbs or on the trunk.

Course and Prognosis.—The attacks when mild may appear at irregular intervals, sometimes several times during the day. When attended by local asphyxia they may occur daily, and then permanently or temporarily disappear, recurrences being brought about by renewed exposure to cold. In the most severe cases in which gangrene develops, death may terminate the process. As a rule, however, Raynaud's disease is not fatal; and although often very prolonged, recovery finally takes place in most instances after numerous recurrences.

Other symptoms may appear in the course of the disease. Repeated attacks may bring about a thickening of the skin of the hands. Hemoglobinuria may accompany the attack, or replace it. Cerebral symptoms, such as loss of consciousness, delirium, temporary hemiplegia, or convulsions, may be seen in some cases, and severe abdominal pain may occur.

Diagnosis.—This is, as a rule, easy, based upon the paroxysmal nature and the symmetrical distribution. Chillblains resemble the syncopal attacks of Raynaud's disease, but do not exhibit a paroxysmal character, and do not so often affect the ultimate terminations of the extremities. Gangrene from other causes is without the symmetrical character, and there is no previous history of milder attacks.

Treatment.—There appears to be no treatment which has any positive action in preventing or relieving the attack. Of most importance is the avoidance of fatigue, local chilling, or sudden changes of temperature. Congenital syphilis should be sought for and treated if present. Galvanism and massage have seemed to be of benefit in some instances. Nitroglycerine is theoretically indicated, and should be tried. Severe pain may require opiates. Immersion in warm water may relieve the local syncope in the milder cases. Any destruction of tissue which occurs demands appropriate surgical measures.

SECTION VII

DISEASES OF THE GENITO-URINARY SYSTEM

CHAPTER I

DISORDERS OF URINARY SECRETION

The characteristics of the urine in disease in childhood, and especially in infancy, is a matter to which too commonly not sufficient attention is given by physicians. It would be advisable to have a routine examination made in every instance, even although this necessitates some trouble. At the least in every case of uncertain diagnosis, or when the course is not proceeding entirely favorably, such examination should be conducted. Many an unexplained condition will become clear if this procedure is followed, and even the saving of life may depend upon our knowledge of the state of the renal secretion. The method of procuring the urine in infancy has already been discussed (Vol. I, p. 192), and the physiological characteristics of the normal secretion of the kidney also studied (Vol. I, p. 60). A certain number of urinary symptoms will be considered here, some of them secondary to primary conditions which will be described independently.

DISTURBANCES IN THE QUANTITY OR THE METHOD OF THE EXCRETION OF URINE

1. Anuria (*Ischuria; oliguria*).—This in the broader sense is the failure to excrete urine in normal amount, if at all; in a narrower sense indicating the latter condition only. It may be divided into (*a*) retention of urine, and (*b*) suppression of urine.

(*a*) **Retention of Urine; Dysuria.**—In this condition the secretion is in normal amount, but there is a failure of the bladder to expel it. In the new born this may be due to some malformation of the urinary apparatus, such as great phimosis or atresia of the labiæ; or to an obstruction in the urethra by uric acid crystals, or a calculus. A balanoposthitis, vulvovaginitis, or inflammation of the meatus may cause retention through the pain which urination produces, especially if the urine is highly acid. Disturbances in the innervation of the bladder are likewise factors; in this class being various reflex or direct inhibitions, such as may occur in meningitis, rectal irritation, myelitis; spasm of the sphincter of the bladder, hysteria, and the debility attending fevers, especially typhoid.

(*b*) **Suppression of Urine.**—Here the urine is secreted in small amount, or temporarily not at all. In the new born 24 hours may sometimes elapse after birth with no urine appearing and the bladder being empty. Uric acid infarcts in the kidney are the active agent in some such cases. Acute nephritis is attended by a diminution in the amount of the secretion. Among other causes of great diminution or suspension of the secretion of urine are profuse watery diarrhea; hysteria; inanition; prostration after surgical operations; and the like.

2. Polyuria.—This consists in a large increase in the amount of uriene passed. In its most characteristic form it is seen in diabetes mellitus and diabetes insipidus (Vol. I, pp 637, 641). It occurs also after the fall of temperature following acute febrile diseases; in interstitial nephritis; after convulsions; and in many children who imbibe an unusual amount of liquid. Nervous excitement and chilling of the surface of the body may produce a temporary great increase in the amount of the secretion. Successful treatment of serous effusions by diuretics and cardiac tonics is attended by polyuria.

3. Frequent Urination (*Pollakiuria*).—This is a normal condition in the first 2 years of life (see Vol. I, p. 61) and is also a necessary symptom of polyuria. It is present in cystitis and occurs from reflex stimulation in the case of renal calculi. A highly concentrated, acid urine causes frequent urination through the irritation of the bladder produced. (See Spasm of the Bladder, p. 205.) It is seen also in any condition of nervous excitement, and is an attendant symptom of diurnal enuresis.

4. Incontinence of Urine.—Apart from the enuresis of childhood, which is to be considered a neurosis and which will be discussed later (see p. 206), incontinence of urine is dependent upon a variety of causes. It is observed, for instance, in phimosis of high degree; impaction of a calculus in the urethra; or in paralytic conditions of the bladder either the result of disease of the brain or spinal cord, or developing in the profound exhaustion or coma occurring in the course of acute febrile or other diseases. Under these circumstances the bladder becomes over-distended with urine until there is a more or less persistent overflow, or, in diseased unconscious states, sometimes an intermittent expulsion of urine without the patient's knowledge. Other cases depend upon various malformations which allow the urine to flow constantly. Such are extrophy of the bladder; abnormal opening of the ureters into the vagina; persistent urachus; and absence of the sphincter vesicæ. Incontinence may accompany, too, severe inflammation of the bladder, as in cystitis, and it is a characteristic symptom in many idiots.

ALTERATIONS IN THE CHARACTER OF THE URINE

1. Albuminuria. (*a*) **Physiological Albuminuria.**—The presence of some of the albuminous bodies in the urine is of very frequent occurrence. Delicate tests show this in the majority of individuals, and even those commonly employed reveal traces in many apparently entirely healthy persons. Hamill and Blackfan[1] found serum-albumin in 27.4 per cent. of 124 normal children, and an albumin precipitated by acetic acid (nucleoalbumin?) in 85.4 per cent. In all, 88.7 per cent. exhibited an albuminuria. The frequent occurrence of albumin in the urine of the new born has already been referred to (Vol. I, p. 61). This may be either nucleoalbumin or serum albumin. Later the presence of small amounts of albumin in the urine may sometimes follow unusually violent exercise, over-eating, cold bathing or indigestion. In these cases the albumin is usually nucleoalbumin, and no granular or epithelial casts or renal epithelium is found in the urine, although occasionally hyaline casts may be present in small numbers. Under these circumstances the condition is a functional disturbance not dependent upon any organic lesion, and possesses no pathological significance.

(*b*) **Orthostatic Albuminuria.**—(*Intermittent Albuminuria; Orthotic Albuminuria; Postural Albuminuria; Cyclic Albuminuria; Lordotic*

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[1] Amer. Jour. Dis. Child., 1911, I, 139.

Albuminuria.) Although this form of albuminuria, described especially by Pavy,[1] is in a sense physiological, since it has no organic renal change as its basis, it presents certain peculiarities which warrant for it a separate consideration.

Etiology.—It is not an uncommon affection; Gőtsky,[2] having found it in 5½ per cent. of one series of 2158 children of from 5 to 14 years, and in 4.4 per cent. of another series of 2031 children. It is seen particularly in later childhood, and with about equal frequency in males and females. In Heubner's[3] series of 56 cases, 22; *i.e.* 39.29 per cent. occurred in individuals of 15 years of age or less. It is uncommon before later childhood. A disposition to manifest itself in certain families has also been noted. Of the various theories proposed to account for its occurrence the prevailing one attributes it to circulatory disturbance, such as produced by the change of position from the recumbent to the upright (*postural albuminuria*), associated with an alteration of the blood-pressure. It should be stated, however, that Bass and Wessler[4] in a study upon a series of cases in children found the blood-pressure differing but little from the normal condition. On account of the influence of lumbar lordosis the disease has been denominated *lordotic albuminuria* by Jehle,[5] pressure being exercised upon the renal vessels by the spinal curvature.

Symptoms.—The characteristic symptom is the appearance of albumin in the urine at certain definite periods of the twenty-four hours. Oftenest it is seen in that secreted during the daytime, but not in that which has accumulated in the bladder during the night. Thus the urine passed on first rising contains no albumin while that excreted in the afternoon shows its presence. The influence of posture is decided. The erect position of the upper portion of the body, especially if the shoulders are thrown back to exaggerate the lordotic curve of the lumbar region, increases the amount of albumin excreted. Hempelmann[6] has found that this produces also a retardation of elimination in the phenolsulphonphthalein test. In normal children it is without influence. The albumin is generally in small amount only. It may be solely an albumin precipitated by acetic acid (nucleo-albumin?), or there may be serum-albumin in addition. No casts are present, except occasionally some of a hyaline nature. Some of the subjects are in apparently perfect health in other respects; more, however, show pallor, digestive disturbances, malaise, faulty general nutrition, or fatigue from slight cause; or nervous symptoms of various sorts including headache, drowsiness, vertigo, a tendency to syncope, and evidences of a neurotic temperament. The blood may not reveal an anemic condition, even in children who are pale.

Course and Prognosis.—The course of the disease is prone to be slow. Sometimes there are intermissions during which albumin disappears completely for weeks or months. The ultimate prognosis is usually considered good, and the disease tends to disappear as adolescence is passed. Langstein[7] and others maintain that the disease never eventuates in nephritis, and may exist for years without doing damage; and that patients who have finally shown the evidences of renal inflammation were in reality suffering from this from the beginning, with a tendency to that intermittent elimination of albumin which is not uncommonly observed in nephritis.

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[1] Lancet, 1885, II, 706.

[2] Jahrb. f. Kinderh., 1910, LXXI, 427.

[3] Henoch's Festschrift, 1890, 170.

[4] Arch. Int. Med., 1914, XIII, 39.

[5] Die lordotische Albuminurie, 1909.

[6] Amer. Jour. Dis. Child., 1915, X, 418.

[7] Pfaundler und Schlossmann, Handbuch der Kinderheilkunde, 1910, IV, 49.

Diagnosis.—The facts mentioned make the diagnosis difficult, since only careful prolonged observation can produce assurance that an intermittent albuminuria is not the manifestation of nephritis in its early state or during recovery. In other respects the differentiation rests upon the absence in orthostatic albuminuria of granular and epithelial casts, enlargement of the heart, edema, and retinitis.

Treatment.—This consists in the improvement of the general health, a defect in which so often underlies the renal condition. No alteration of the diet is required, except such as may aid in the removal of any attending dyspeptic symptoms. Anemia demands treatment if present. A moderate amount of exercise is permissible, but that of a violent nature is to be forbidden, as is prolonged standing. The advisability of rest in bed during a portion of each day is disputed. Abundant out-door life in a favorable climate is of great importance. Efforts should be made to correct a lordosis present by proper postural and gymnastic treatment; but the wisdom of wearing an apparatus for this purpose is questionable.

(*c*) **Febrile Albuminuria.**—The exact nature of this disorder is not definitely determined, some believing that the cloudy swelling of the cells of the glomeruli, which appears to be the cause, should be considered as a pathological lesion; others denying this. In any event it cannot properly be designated a nephritis, since the element of inflammation is wanting. The majority of cases of acute infectious diseases with continued fever exhibit small amounts of albumin in the urine; and, in fact, elevation of temperature from whatever cause is liable to have this result. On the cessation of the fever the albuminuria disappears. Hyaline casts and even an occasional epithelial cast may be present. (See Acute Degeneration of the Kidneys, p. 175).

(*d*) **Adventitious Albuminuria.**—Only the reminder need be given here that the presence in the urine of blood or of pus from any source will produce an albuminuria. Microscopical examination reveals the cause.

(*e*) **Albuminuria from Organic Renal Disease.**—This variety will be considered later under the respective headings of the different diseases of the kidneys.

2. Pyuria.—This is characterized by the appearance in the urine of pus-corpuscles, at least 6 to 8 to every field of uncentrifuged urine, generally with epithelial cells from the bladder or the pelvis of the kidney. If the pus is abundant the urine will show the presence of albumin. There is a whitish or yellowish-white, dense sediment, and the supernatant urine is turbid. Pyuria may be produced by various causes and may be either acute or chronic. When dependent upon a vulvo-vaginitis the pus is present usually in small amount. Pyuria resulting from an inflammation of the urethra is uncommon in early life. Cystitis gives rise to pyuria generally with an admixture of mucus; the urine being either acid or alkaline and the pus usually not in large amount. Oftenest in children it is acid, since infection by the colon bacillus is the most frequent cause at this period of life. The commonest source of pyuria in children is inflammation of the pelvis of the kidney. (See p. 196.) It may also be the result of renal calculi, tuberculosis of the kidney, or pyonephrosis. In all these the urine is acid and the pus often present in large amount. Acute nephritis exhibits scattered pus-cells with epithelial casts in the urine.

Sometimes an unusually large quantity of pus is found in the urine, but soon nearly or entirely disappears. This is the case when an abscess from without the kidney, as of a perinephritis, perityphilitis, or of other nature, ruptures into the urinary tract.

The treatment of pyuria depends upon the cause.

3. Bacteruria. Bacilluria.—Although numerous microörganisms are found in most cases of pyuria, the term is more conveniently applied to those cases in which there is an excretion of bacteria in unusual numbers without the presence of pus and generally without constitutional symptoms. The urine is cloudy from the great quantity of germs present, usually colon bacilli. This disease is seen at any period of early life, oftenest in females, but seems to be an uncommon one. Mellin[1] found but 11 cases in medical literature to which he added 10 of his own; this large addition indicating that a systematic examination of the urine would show the disorder more frequent than has been supposed. It may be an early symptom of pyelitis or cystitis and require the treatment suitable for these.

4. Hematuria.—Red blood-cells may appear in the urine either in such small numbers that only microscopic examination reveals them, or in such large amount that the urine is of a blood-red or a brown color. Intermediate between these is the urine with sufficient blood to give it a characteristic "smoky" appearance. The **causes** are various. In infancy hematuria may be evidence of the hemorrhagic disease of the new born, acute nephritis, scurvy, tumors of the kidney, sepsis, or uric acid infarction. Later it is a symptom of calculus in the kidney or elsewhere in the urinary tract, purpura, hemorrhagic forms of the acute infectious disorders, nephritis, tumors of the kidney or bladder, tuberculosis of the kidney, severe passive renal congestion, hemophilia, leukemia, bilharzia, trauma, and the action of irritant poisons.

An idiopathic hematuria of unknown origin has been observed in rare instances. It is congenital in its development. Guthrie[2] reported 12 instances in 2 generations of a single family. The bleeding may be persistent or may come on in recurrent attacks lasting a few days or weeks, and the entire process may continue perhaps some years. Slight constitutional symptoms may be present.

The determination of the seat of hemorrhage in hematuria is often very difficult. The presence of blood-casts is proof of the renal origin, but their absence is of no diagnostic value. Large hemorrhages are generally from the kidney. Cystoscopic examination at the time of hemorrhage may show from what region the blood is coming. If the blood appears only at the beginning or the end of micturition it is probably from the lower urinary tract.

The **diagnosis** of hematuria can be made certain only by microscopic examination and the discovery of red blood-corpuscles. Not infrequently in infancy urine is passed which stains the diaper a slightly red color, dependent upon urates or the coloring matter of the urine, but which is often supposed to be due to blood.

The **prognosis** depends upon the etiology and the **treatment** is chiefly that of the cause. In addition the child should be kept at rest in bed and given a light diet, and injections of horse-serum may be administered if the hemorrhage is severe.

5. Hemoglobinuria.—The urine in this disorder exhibits the coloring matter of the blood, but with very few red blood corpuscles or none at all. Albumin is generally present in small amount, and there is a brownish precipitate which under the microscope is seen to consist of granular pigment. Hyaline and granular casts are usually found. The urine macroscopically is red or reddish-brown in color and sometimes almost black. That the color depends upon hemoglobin or methemoglobin may be shown by Heller's test or by the spectroscope.

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[1] Jahrb. f. Kinderh., 1903, LVIII, 40.

[2] Lancet, 1902, I, 1243.

Hemoglobinuria may be seen in severe infectious diseases; be one of the results of burns; be produced by certain parasites; or depend upon the action of certain mineral or other poisons such as carbolic acid, chlorate of potash, oxalic acid, arsenic, phosphorus, and quinine. Hemoglobinuria of the new born has already been described (Vol. I, p. 262). A rare condition in children is that entitled "**paroxysmal hemoglobinuria.**" According to Herrman[1] only about 24 cases have been published occurring at this period of life. The cause of this variety is not certainly known, but the action is clearly a hemolytic one occurring at the time of the attack, and the resistance of the red cells is diminished (Reiss).[2]

Hemoglobinuria may occur either in infancy or childhood. Some cases appear to depend upon chilling of the surface of the body; others on over-exertion; others on syphilis. In 77 reported cases occurring at different periods of life Stempel[3] found syphilis present in 29.8 per cent. The passing of hemoglobin may be preceded by chilliness, fever, digestive symptoms, prostration, and sometimes headache or pain in the lumbar region. In some instances there are evidences of vasomotor spasm, such as cyanosis, coldness of the extremities and pallor. Other cases have no symptoms except the urinary ones. The discharge of hemoglobin may last from some hours to 1 or 2 days.

The **prognosis** of hemoglobinuria depends upon the cause. In the paroxysmal variety the disease itself is usually not dangerous, except through the development of complications, chiefly nephritis. **Treatment** consists in rest and warmth, and especially of residence in a warm climate. Antisyphilitic remedies are indicated in some cases.

6. Glycosuria. (*Lactosuria, etc.*).—Sugar in some form is not infrequently found in very small amount in the urine of apparently healthy infants in the early months of life. The appearance of traces of lactose in the urine of healthy breast-fed or bottle-fed infants can scarcely be called pathological. In infants with gastrointestinal diseases there is an increased tendency for lactose to pass the intestinal wall unchanged and appear in the urine. Gaujoux[4] found that the feeding of large amounts of saccharose to normal infants was capable of producing a transient saccharosuria in them, sometimes with glucose in small amount as well. The temporary presence of a small quantity of glucose in the urine in healthy children given unusually large amounts of sugar, or sometimes of starchy food, is termed *alimentary glycosuria.* A transient glycosuria may sometimes result, too, from the action of such poisons as mercury, opium, or carbon dioxide; may at times occur in the course of the infectious diseases, or of disorders interfering with respiration, such as pertussis and pneumonia; or may be attendant upon certain nervous maladies, as convulsions, meningitis, intracranial tumors, or injury from fracture of the skull. The continued excretion of a large amount of sugar is seen in diabetes. An uncommon condition at any period of life is that in which the form of sugar called pentose is found in the urine (*pentosuria*). It is important only in that it must be differentiated from diabetes. It does not respond to the fermentation test. In childhood it is rare. Aron[1] reported an instance in a boy of 5 years.

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[1] Arch. of Ped., 1903, XX, 105.
[2] Jahrb. f. Kinderh., 1913, LXXVIII, 723.
[3] Centralbl. f. d. Grenzgebiete d. Med. u. Chirurg., 1902, V, 184.
[4] Ann. de méd. et de chir. inf., 1913, XVII, 129.

It is of particular importance in determining the existence of sugar in the urine to remember that other reducing bodies may be present. The copper-test alone must, therefore, not be depended upon, and the fermentation-test or that with phenylhydracin be used or the polariscope employed. Further, too, is to be remembered that to constitute diabetes there must be the constant presence of a considerable amount of glucose in the urine and that the occurrence of sugar of other nature is not a symptom of the disease.

7. Lithuria; Uric Acid Infarcts.—Lithuria, or the excretion of uric acid or its salts in abnormal amount, is found in early life under several conditions. The cause is either an insufficient secretion of water by the kidneys, or an over-production of uric acid or of urates. Children of any age with indigestion are liable to an excess of it. In infancy the condition may show itself in the form of a whitish or reddish deposit upon the diaper. The presence of fever, too, increases the percentage of uric acid excreted, partly probably through its increased production and partly through concentration of the urine. In the new born the amount of water in the urine may be so diminished that a deposit of uric acid or urates takes place in the straight tubules of the kidney producing *uric acid infarctions*. A slight development of this condition is very common. Generally the crystals are washed out in the early weeks of life as the secretion of water becomes more free. In passing they may give rise to priapism and to severe pain, shown by attacks supposed to be ordinary intestinal colic. The evidence of the passage may be seen upon the diaper. In the treatment of lithuria the administration of water freely, preferably alkalinized by the addition of citrate of potash, 8 to 10 grains (0.52 to 0.65) during the 24 hours, aids in relieving the condition.

It is to be borne in mind that an excess of uric acid cannot be determined by the mere discovery of a precipitate in the urine, since various conditions may make the urates on this occasion more insoluble than usual; nor by the chemical analysis of a small quantity of the fluid from a single passage. A high acidity of the urine may readily account for the precipitate.

8. Indicanuria.—Indican is generally absent from the urine in healthy breast-fed infants, but present in small amount in those artificially fed. When in larger quantity it usually depends upon digestive disturbancs in which abnormal putrefaction of proteid matter in the intestine is taking place. It is a frequent attendant upon constipation. It may also less commonly result from decomposition of protein occurring in other parts of the body. The usual indications for treatment are to alter the diet in such a way as to hinder abnormal intestinal changes, especially by diminishing the amount of protein ingested.

9. Lipuria.—This is characterized by the appearance of fat-globules in the urine; guarding against mistaking for this the adventitious presence of fat in the vessels into which the urine is passed, or present on a catheter which had been employed. The existence of fat in the urine may depend upon an excessive amount of fat in the food, or upon fatty degeneration of the kidneys. It is also seen typically in **chyluria**, in which the fat is present in sufficiently large amount to give the urine a milky appearance.

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[1] Monatsschr. f. Kinderh., Orig., 1913, XII, 177.

Albumin also is always found in chyluria and sometimes red blood-cells as well. The course is chronic and the prognosis uncertain. Some cases are due to the presence of filaria, which may be discovered in the blood. Non-parasitic chyluria is a rare affection in early life. Hymanson[1] reported a case in a boy of 8 years, and could find only 5 others which could be classed in any way as occurring in childhood.

10. Acetonuria.—The various acetone-bodies, acetone, aceto-acetic acid, and β-oxybutyric acid, occur in the urine under various conditions, but the cause in all is a disturbance of metabolism, especially of that of the fat, although other elements may share in the process. The lack of ingestion or lack of assimilation of the carbohydrates is the cause of the faulty fat-metabolism. (See Vol. I, p. 49.) Acetonuria is especially well seen in diabetes and in the recurrent vomiting of children. (See Vol. I, pp. 639 and 702.) It occurs also in acute gastrointestinal diseases and acute febrile disorders, especially pneumonia and the infectious fevers, and in many conditions of autointoxication of intestinal origin, and it appears in all hunger-states when no food is taken or retained. It is to be clearly distinguished from Acidosis. (See Vol. I, p. 635.)

11. Urobilinuria.—Urobilin in the urine in small amount has no special significance. It is not found in the case of breast-fed children, but is often present in the bottle-fed. It may occur in increased quantity in those with intestinal disorders, pernicious anemia, infantile scurvy, some febrile infectious diseases, and in hemorrhagic disorders.

12. Alkaptonuria.—This is an unusual congenital condition, sometimes familial, and occasionally directly transmitted. It appears in early infancy and lasts indefinitely. The characteristic is the passage of urine which is at first normal in color, but which on exposure to air becomes brown or deep black. This change depends upon the presence of homogentesic acid or allied aromatic substances. Alkaptonuria appears to have no influence whatever upon the general health. Sometimes in later life chronic affections of the joints have been found associated with it.

13. Cystinuria.—In this disease, occasionally observed, which is often familial and hereditary and in most cases congenital, cystin is found in the urine in the form of the characteristic plates, or may be retained in the bladder and form a calculus. Except for the danger of the latter, the anomaly appears to exert no influence upon the general health.

14. The Diazo Reaction.—This is not present in the urine of healthy breast-fed infants, nor is it produced in the course of the majority of febrile or other diseases. It is found constantly, however, in measles, typhoid fever, and most forms of tuberculosis, and it may occur in some cases of other infectious disorders.

15. Abnormally Colored Urine.—Occasionally the abnormal color of the urine attracts attention. Apart from the red tint due to blood; the reddish-yellow color from the presence of bile, and the discoloration produced by reddish urates, we may find certain tints, the result of the action of drugs or other chemical substances. Few of these are liable to be causative factors in early life, except those present in some of the artificially colored candies. Thus a green or blue urine has been produced by the ingestion of methylene blue; purple or red by fuchsin; a magenta by phenolphthalein; a red with a greenish fluorescence by eosine. Black urine may be a symptom of carbolic acid poisoning.

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[1] Amer. Jour. Dis. Child., 1916, XI, 455.

16. Ammoniacal Urine.—Not infrequently the urine of an infant smells strongly of ammonia after it is evacuated. If considerable time has elapsed and decomposition is well under way this characteristic is a matter of no moment; but in the cases where a strong odor of ammonia develops soon after the emptying of the bladder the indication is that there is a disturbance of the metabolism present, and that an excess of acid has taken from the tissues the available fixed alkali and necessitated a combination of the remaining acid with the ammonia, instead of the usual transformation of this into urea being consummated. The condition is often brought about by the excessive formation of fatty acids, dependent oftenest upon a malassimilation of the fat of the food. The therapeutic indications are to administer alkalies, which will prevent the abnormal combination and the robbing of the tissues of the fixed alkali; and at the same time to order the diet appropriate, to the intent that an excess of acids shall not be produced (Southworth).[1]

CHAPTER II

DISEASES OF THE KIDNEYS

MALFORMATIONS OF THE KIDNEYS

Congenital malformations of the kidneys are seen with comparative frequency and are of various forms. The lobulated kidney, characteristic of fetal life, may be present to an unusual degree and persist into childhood. It is of no clinical significance. The kidneys may be entirely absent; or that upon one side, especially the left, may be absent or imperfectly developed, that upon the other then being correspondingly enlarged. The kidneys may be fused into a mass of various forms, the horseshoe kidney being the most frequent. Malformations of the ureter frequently accompany those of the kidneys.

The various renal anomalies have been reviewed by Rolleston.[2] They are of little clinical importance, with the exception of the rare absence of the kidney, or absence or closure of the ureters, both of which are incompatible with life. There are two, however, which must receive separate consideration.

Congenital Cystic Kidney.—In this condition the kidneys undergo a cystic transformation, the parenchyma being occupied by small or larger cysts, sometimes very numerous. Usually both kidneys are involved, although perhaps to different degrees. They may not be increased in size or, on the other hand, may become very large, even in fetal life, and then may interfere with birth. In cases seen later the size may be found to increase under observation.

The cysts are the result of occlusion, but the manner in which this is brought about is disputed. Other anomalies are frequently present elsewhere in the body. The patient often dies in early infancy with the symptoms of uremia; but if sufficient secreting tissue remain may live to adult life. The principal symptom in the case of large cystic kidney is the increase in size of the abdomen, with the discovery of a tumor in the renal region. Removal of the kidney is to be performed only in the uncommon cases where it has been proven that the condition is unilateral. I have known death to follow the neglect of this precaution.

[1] Arch. of Ped., 1913, XXX, 732.

[2] Brit. Jour. Child. Dis., 1916, XIII, 80.

Hydronephrosis.—This is not uncommon in early life, and may be either congenital or, less often, acquired. Although in the latter event it is, strictly speaking, not a malformation, it may conveniently be included in this description. It consists in a dilatation of the pelvis of the kidney and often of the ureter, with consecutive atrophy of more or less of the renal parenchyma. The disease is produced by some obstruction to the passage of the urine. This may occur anywhere in the course of the ureter, bladder, or urethra; such as an impacted calculus, organic stenosis, external pressure, extreme phimosis, and the like; but the cause is not always discoverable even at autopsy, and in such cases may depend upon an intra-uterine defective coördination of the nervous and muscular control of the excretory apparatus (Thomson).[1] Severe trauma of the kidney is sometimes followed by a hydronephrosis. Of this occurrence Aldibert[2] collected 17 instances. Hydronephrosis may involve one or both kidneys. It may remain small, or may become so large that a fluctuating abdominal tumor is produced, sometimes of great size, and occupying the renal region or filling the greater part of the abdominal cavity. In the congenital cases the distention may be so great that labor is interfered with. In these cases malformations of other parts of the body may be present. Aspiration of a hydronephrosis reveals a fluid which examination shows has the characteristics of urine. When the condition is bilateral and well-marked life cannot be long-continued. The patient develops the evidences of chronic nephritis and dies from uremia or from some intercurrent disease, usually in infancy.

Unilateral hydronephrosis gives no signs until the sac is large enough to be discovered by physical examination. Later the evidences of chronic nephritis may appear.

Treatment is possible only in cases of unilateral hydronephrosis and is entirely surgical. The cyst may be incised and drained, but as the development of pyonephrosis is a very possible danger, usually nephrectomy is a preferable operation.

DISPLACEMENT OF THE KIDNEY

Dystopia.—Some of the malformations described, especially the horseshoe kidney, show displacement also. In other cases the kidney may be displaced without any accompanying malformation, and is usually fixed in the abnormal position. It is liable to occupy a situation lower than normal, and nearer the middle line. That upon the left side is oftenest involved. Sometimes the ectopic kidney is recognized as a small tumor, and confusion with other conditions readily arises, notably intussusception, fecal accumulation, appendicitis, and undescended testicle. There may be no subjective symptoms whatever; or, should the ureters be pressed upon or bent, dysuria and pain in the lumbar region and lower limbs may develop.

Movable Kidney. **Etiology.**—This is a form of renal displacement requiring special consideration. Although not common in early life it is probably more frequent than often supposed. It is usually upon the right side. Comby[3] observed 18 cases in children, 16 of which were in girls; these ranging in age from 1 month up to 10 years. The condition may be congenital or acquired. The acquired cases are oftenest seen toward the end of later childhood. Dupoux[1] could collect but 38 instances of this sort in children, 31 of these being in girls. In this variety it may be the result of the pressure of tumors or corsets, trauma, and digestive disturbances. When of congenital origin it depends upon an unusually long pedicle. Phillips,[2] Comby,[3] and others, have reported its discovery in the new born.

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[1] Brit. Med. Jour., 1902, II, 678.
[2] Rev. men. des mal. de l'enf., 1893, XI, 441.
[3] Pediatrics, 1898, VI, 439.

Symptoms.—No symptoms whatever may be observed, or they may be of an indefinite nature, such as dyspeptic disturbances, nervousness, and a sensation of pressure and dragging in the abdomen or the lumbar region. The most characteristic symptom is the occurrence of attacks of pain, sometimes brought on by exercise. This is of the nature of colic and may radiate into the extremities; but it is rarely as severe or as often witnessed as in adults. The symptoms of general splanchnoptosis may be present also, since this condition is frequently combined with movable kidney. A positive diagnosis can be made only by the discovery by palpation of a smooth movable body having the general shape of the kidney and capable of being readily pushed into the normal position for this organ. The mere ability to palpate the lower end of the kidney is not sufficient for diagnosis, since Baron[4] examined 283 children under 15 years of age and was able to palpate the kidney in 83.

Treatment.—This consists in efforts to retain the kidney in position by the use of a pad or bandage, the avoidance of violent exercise, and measures directed to the building up and sustaining of the general health, with careful attention to the digestive apparatus, especially the relief of constipation. Surgical aid will seldom be required in early life.

CONGESTION, DEGENERATION AND INFLAMMATION OF THE KIDNEY

In the discussion of these topics the classification of Delafield and Prudden[5] has been followed for the most part, with such modifications as the early age of the patients necessitates. I am largely indebted to these authors and to Senator[6] for much of the description of the pathological anatomy.

ACUTE CONGESTION OF THE KIDNEYS

Etiology.—The disease may be an early stage of nephritis, or, without this as a sequel, may be the result of fever, exposure to cold, overexertion, surgical operations, trauma, and the poisonous action of such substances as turpentine and cantharides.

Pathological Anatomy.—The condition is characterized by the presence of congestion with the absence of evidences of nephritis or other renal change. The kidneys are swollen, soft, and dark from the overfilling of the vessels with blood.

Symptoms.—Chief among these is the diminution or even suppression of the secretion of urine, which is high colored, and sometimes contains blood-cells, and perhaps albumin in large amount; while tube-casts, generally in small numbers, may be present.

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[1] Thèse de Paris, 1902.

[2] Lancet, 1903, I, 731.

[3] Traité des mal. de l'enf., 1904, II, 712.

[4] Festschrift z. fünzigj. Bestehen d. Stadtkrankenh. zu Dresden. Ref., Jahrb. f. Kinderh., 1900, LI, 714.

[5] Text-book of Pathology, 1911, 746; also Delafield, Transac. Assoc. Amer. Phys., 1891, VI, 124.

[6] Nothnagel's Encyclopedia of Practical Medicine, 1905, Diseases of the Kidneys, 148 et seq.

The **course** is usually short and recovery follows except in cases occurring as the result of surgical operations or injury, or as a complication of renal disease previously existing. Long continuance or great intensity of the cause of acute congestion may lead finally to nephritis. The **diagnosis** from nephritis is made by the short duration and the absence of dropsy.

Treatment.—The patient must be at rest in bed; the skin kept warm; the bowels purged, and diuretics and diaphoretics given. Water should be administered freely, while milk constitutes the best diet. Warm tub-baths, hot packs, hot compresses over the kidneys, and large enemata of warm normal salt-solution are of service in relieving the congestion and reëstablishing the renal function.

PASSIVE CONGESTION OF THE KIDNEYS

Etiology.—This disorder may result from any cause which produces general venous stasis, the most frequent being chronic disease of the heart. It results, too, from intra-abdominal pressure by tumors and from chronic affections of the lungs and chronic pleurisy, and may complicate a nephritis already present. It is seen oftenest in older children.

Pathological Anatomy.—The kidneys are enlarged, hard, of a deep-red color, and bleed freely when incised. The capillary blood-vessels, especially those of the glomeruli, are dilated, engorged with blood, and often have their walls thickened. The capsule of the kidney is not thickened, and is readily stripped from the cortex; the stellate veins beneath the capsule are very prominent, and those of the pyramids are dark-red in color. On microscopical examination the epithelium of the tubules is found swollen or flattened. Should the process be long continued, degenerative and inflammatory changes follow and chronic nephritis results.

Symptoms.—The principal symptoms observed are those of the primary disease which is the causative factor; and it is only by the character of the urinary secretion that the diagnosis of passive congestion of the kidney can be established. The urine is diminished in quantity, high colored, and of a high specific gravity. Albumin is present in small amount only, and at times not at all. Hyaline casts may be discovered; granular casts are more unusual; and red blood-cells, if present, are very few in number. The urinary constituents are increased relatively, but their total output is normal or only slightly below this.

Diagnosis.—The diagnosis of the condition from chronic nephritis is often difficult, especially as the latter is a frequent sequel to any much prolonged renal congestion. The presence of a considerable amount of albumin and of numerous granular casts indicates that nephritis has developed. If improvement in the general symptoms takes place under rest and medication; the albumin disappears; the secretion increases; and the high specific gravity lessens, the condition is almost certainly passive congestion and not nephritis. On the other hand in a long-continued case without alteration for the better in the general symptoms, an apparent improvement in the character of the urine is not a favorable indication, and the change to a pale color with low specific gravity and perhaps increase in amount indicates the probable development of a contracted kidney.

Treatment.—This is necessarily chiefly that of the cause. In addition measures to increase the secretion of urine should be employed, such as the application of dry cups or hot fomentations over the kidneys and the administration of such diuretics as citrate or acetate of potash or theocine.

ACUTE DEGENERATION OF THE KIDNEYS

(Febrile Albuminuria)

Etiology.—The disorder is always secondary, by far the most frequent cause being the existence of pneumonia, rheumatism, septicemia, or one of the acute infectious fevers; the toxins produced by these diseases affecting the epithelial cells of the tubules. In fact the existence of high temperature from any cause, if prolonged, may produce it. It may also be brought about by such drugs or poisons as turpentine, phosphorus, arsenic and cantharides. The condition is extremely frequent in early life.

Pathological Anatomy.—The degree of change varies with the intensity of the action of the poison. The kidneys may be slightly enlarged, and on section the cortex is pale and thickened and the ordinary cortical markings obliterated. The capsule of the kidney can be stripped off readily. Microscopically there are seen all grades of cloudy swelling, from simple granular changes of the epithelium, especially of the convoluted tubules, to the development in the cells of fat-globules or to shedding of the necrotic epithelium. As recovery progresses a growth of new epithelium takes place. In many cases the lesions of inflammation are added to those of degeneration and a true nephritis results. Degenerative changes in the cells of other organs of the body are generally present also.

Symptoms.—In the degeneration attending the febrile diseases there are no characteristic symptoms other than those connected with the condition of the urine. (See Febrile Albuminuria, p. 166.) This is high colored, acid, and contains a small amount of albumin which is chiefly nucleo-albumin, and a few casts, generally hyaline, sometimes epithelial, less often granular; while scattered leucocytes, epithelial cells and red blood-corpuscles may be seen.

Course and Prognosis.—After the fever has ceased the urine resumes its normal character, usually with promptness, although it may occasionally be weeks or months before all traces of the pathological process have disappeared. In this latter event the suspicion of the existence of a nephritis is justified. It is only in the unusual cases dependent upon poisonous substances of other nature than the toxin of fever that the degeneration is so extreme as to cause death.

Diagnosis.—The line between degeneration and true nephritis cannot be clearly drawn; in fact many writers place all these cases in the category of nephritis. To this fact is to be attributed the great variation in the published statistics regarding the frequency of nephritis in infectious diseases, particularly scarlet fever. (See Scarlet Fever, Vol. I, p. 325.) The differentiation rests upon the presence in acute degeneration of but a small amount of albumin and but few, if any, casts; and upon the prompt disappearance of these symptoms with the cessation of fever. In addition the edema or cardiac changes indicative of Bright's disease do not occur.

Treatment.—None is required, and there is none which certainly modifies the process. (See Scarlet Fever, Vol. I, p. 325.) Mild diuretics should be employed and a liquid diet continued in order to throw as little work upon the kidneys as possible.

NEPHRITIS

Many methods of classification of the varieties of this disease have been adopted, with much resulting confusion and a multiplicity of titles. A division solely upon an etiological basis is impossible; while one purely of an anatomical nature is unsatisfactory from a clinical point of view. Moreover the different anatomical conditions usually exist in various combinations, or may be but different stages in the course of the disease. Any classification can therefore but be to an extent artificial and only for convenience of study.

In general, nephritis may be defined as an inflammatory process which is either of an *exudative* character (infiltration by serum and by leucocytes), or *productive* in nature (over-growth of connective tissue and epithelial cells of the Malpighian bodies), and to those changes a *degenerative* process is added (Delafield and Prudden).[1]

ACUTE DIFFUSE NEPHRITIS

(Acute Exudative Nephrtis; Acute Tubular Nephritis; Acute Desquamative Nephritis; Glomerulo-nephritis; Acute Interstitial Nephritis)

Etiology.—Age is not a factor of great importance. Although more common in early childhood, infants are much oftener attacked than is generally supposed. Mensi[2] reported on 17 cases seen in infants of from 10 to 40 days old; and its occurrence in the new born had been previously described by Jacobi.[3] Goulkewitch[4] found evidences of nephritis present in 22 of 220 autopsies on infants of the nursing period. In infancy acute nephritis is oftenest produced by hereditary syphilis, widespread cutaneous eruptions, bronchopneumonia, the acute infectious fevers, and especially sepsis and acute gastrointestinal diseases. The influence of syphilis appears to be not very frequent, but still is undoubted. In 31 syphilitic infants of from 2 weeks to 7 months of age Cassell[5] found nephritis in 6, and cases have been reported by Audeoud,[6] Carpenter[7] and others. I have seen at least 1 instance (Griffith and Newcomet).[8] After the period of infancy acute nephritis is oftenest a distinctly secondary disorder, most frequently seen in the course of or after pneumonia, sepsis, and the acute infectious disorders. Oftenest it is observed in connection with scarlet fever, but there is probably no one of the diseases of this class which may not sometimes exhibit it. Such mild affections as varicella and mumps may occasionally have nephritis as a complication, and it has even been reported after vaccination. Measles is stated to be rarely a cause, yet undoubtedly is operative oftener than supposed. I[9] have seen a number of instances of nephritis dependent upon it. Rheumatism, tonsillitis, diseases of the skin, and meningitis are also causes. I have observed it on several occasions follow operation upon the tonsils and adenoid growths. The influence of chilling of the body has been much discussed, but there certainly are numerous cases which have succeeded immediately upon exposure to cold and wet, without any other discoverable cause. The ingestion of certain toxic drugs, as chlorate of potash, turpentine, carbolic acid and the like, may produce nephritis. Not infrequently the disease occurs without there being any cause discoverable. To such cases the title "primary" has been applied. Many of these are doubtless secondary to some other unrecognized condition, but others appear to be truly primary. In infancy a primary nephritis is quite uncommon, but I have encountered a number of cases which appeared to belong to this class. In older children, too, it is of comparatively infrequent occurrence.

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[1] Text-book of Pathology, 1911, 754.
[2] Riv. clin. pediat., 1903, I, 505.
[3] New York Med. Jour., 1896, LXIII, 65.
[4] Rev. men. des mal. de l'enf., 1900, XVIII, 308.
[5] Berl. klin. Woch., 1904, XLI, 558.
[6] Rev. méd. de la Suisse romande, 1896, XVI, 381.
[7] Brit. Jour. Child. Dis., 1907, IV, 421.
[8] Med. News, 1897, Oct. 2.
[9] Penna. Med. Jour., 1908, XI, 971.

The direct cause of the nephritis is usually either the toxins produced by bacteria or in other ways, or the action of the bacteria themselves. There are cases, however, which do not appear to come under either category.

Pathological Anatomy.—The lesions vary with the stage of the nephritis, with the case, and to some extent with the cause. The kidney is larger and softer than normal; the capsule readily removable. On section the cortex is thickened and sometimes pale, sometimes reddened; sometimes mottled in both colors as the result of the presence of scattered collections of blood. Often the pyramids may seem unusually red in contrast with the pale, thickened and edematous cortex. The Malpighian bodies stand out as dark points. Microscopically all the tissues of the kidney are found involved, but in a varying degree and not uniformally throughout the kidney. The glomeruli exhibit swelling, and sometimes proliferation and alteration in the form of the cells covering the tufts and lining Bowman's capsule. These cells may undergo fatty degeneration and exfoliation, and, combined with albuminous exudate, leucocytes, and red blood-cells, sometimes largely fill the capsule and may compress the tuft. The tubules of the kidney are also involved, the changes being chiefly degenerative. The epithelium, especially of the convoluted tubules, is swollen and degenerated and contains granular matter and some fat-globules. It may be exfoliated, and, with albuminous granules, leucocytes and red blood-cells, may form in the tubules of the cortex or of the pyramids casts of various sorts, the nature of which depends upon the predominence of one or another of these substances. The interstitial tissue suffers also, there being at first edema and then an infiltration with round cells and fibrinous exudate, and finally, if the case continues longer, the production of new-formed connective tissue.

As already stated the degree of these different changes varies. To the condition in which the alterations are principally in the glomeruli, the title *glomerulo-nephritis* has been applied. When the change is particularly in the epithelium of the tubules with little exudation, it is entitled *parenchymatous nephritis* or *degenerative nephritis*. This is the form especially well-marked early in severe cases of diphtheria, and is that seen in gastro-enteric diseases in infants. When there is considerable hemorrhage into different parts of the kidney, the title *hemorrhagic nephritis* has been employed. To the form in which exudation of plasma and of leucocytes into the Malpighian bodies, the interstitial connective tissue, and the tubules predominates, the term *exudative nephritis* has been applied by Delafield.[1] On the other hand, the condition in which proliferation of the capsule-cells of the Malpighian bodies and the formation of new connective tissue in the kidney are the chief histological changes, the same author has called *productive nephritis*, also frequently denominated *interstitial nephritis*. This variety is oftenest seen after long-continued severe cases of diphtheria or especially of scarlet fever, or may occur as an independent process.

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[1] Transac. Assoc. Amer. Phys., 1891, VI, 128.

These different conditions must be considered as mere varieties of one nephritic process. They may be combined in different degrees in the same case and various intermediate forms occur.

Symptoms.—The nephritis occurring in connection with scarlet fever has already been discussed (Vol. I, p. 325), and the description applies to some extent to the disease dependent upon other causes. The symptoms are subject to great variation, and often are masked by those of the primary disorder. They differ in intensity, too, with the severity of the inflammation, and to some extent with the age of the patient. In typical cases the onset is sudden, with fever of varying degree (Fig. 316). Some degree of edema is frequent, especially about the face and ankles; but a well-marked development of it is less common. Occasionally, however, there may be anasarca, and even effusion into the lungs and serous cavities, or into the tissues of the larynx producing edema of the glottis. On the other hand, edema may be entirely absent. Anemia rapidly develops as the disease progresses. The blood-pressure is increased, and especially so when edema is absent, as in the hemorrhagic cases. It may reach as high as 180 mm. systolic pressure (Gordon).[1] Symptoms apparently uremic are not common in nephritis in early life, but occasionally are marked and may be the earliest manifestations; expressed by headache, vomiting, sometimes diarrhea, dyspnea, dullness, coma, or convulsions. It is very possible, however, that these symptoms are dependent upon an intoxication of other nature, perhaps an acidosis.

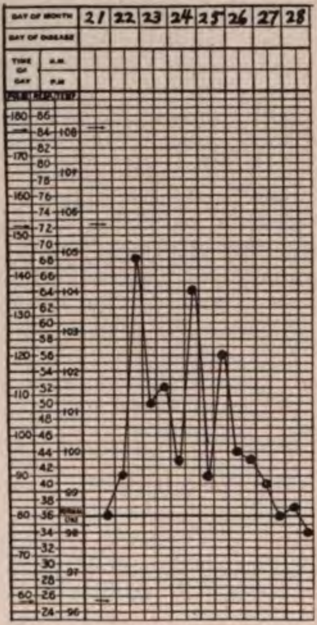

FIG. 316.—ACUTE NEPHRITIS.

Katie G., 4 years old. Taken ill Feb. 19, edema of feet, face and arms. Entered the Children's Hospital of Philadelphia, Feb. 21, face much swollen, could not open right eye, urine very much diminished and contained hyaline and granular casts, red blood-cells and leucocytes, nothing found in lungs, respiration somewhat accelerated, gradual improvement.

In infancy especially the symptoms are often obscure and overlooked, edema may be absent, and only an examination of the urine reveals the true nature of the malady; although even albuminuria may be absent for a time, as shown by Cassell,[2] Quincke,[3] Dickinson[4] and others. Latent cases of this sort are perhaps oftenest seen in connection with gastroenteric disorders. In older children, too, the onset is often insidious. There may be no characteristic evidences of nephritis, and the diagnosis is impossible except by urinalysis. The urine may be but little diminished in amount in the milder cases, and even in some of the severer ones; but, as a rule, in well-marked cases it is decidedly lessened in quantity; acid; and either pale or of a smoky, dark-brown, or bright-red color from the presence of blood. The specific gravity is of little moment. It can be high in mild cases, or possibly quite low in severe ones, even with little secretion of urine. Occasionally the secretion is almost or quite suppressed, but this is not common and generally occurs only when decided symptoms of uremia are present. I have, however, observed entire suppression for several days, with the existence of uremic symptoms very doubtful. As a rule the diminution of the urinary secretion and its alteration in appearance are in proportion to the severity of the case, but there is no certain relation between the state of the urine and the degree of edema. Albumin is present in varying amount; sometimes only a trace, oftener abundant; sometimes so much that the urine solidifies on boiling. The quantity of urea excreted is decidedly lessened. Under the microscope there are found casts of diverse sorts including those of an epithelial or granular type. Red blood-cells, leucocytes or renal epithelium may be present or absent, depending upon the nature of the predominating lesion. Some blood is nearly always found at the beginning of the attack.

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[1] Arch. of Ped., 1911, XXVIII, 343.
[2] Berl. klin. Woch., 1904, XXXVII, 213.
[3] Berl. klin. Woch., 1882, XIX, 57.
[4] Transac. Path. Soc. London, 1870, XXI, 255.

The symptoms depend somewhat upon the primary cause. The most typical complex is best illustrated in the nephritis occurring about the end of the 3d week after the onset of an attack of scarlet fever. Some degree of edema is nearly always present in this condition. Diphtheritic nephritis may be mild or severe and suppression of urine occur; but edema is less often seen. Nephritis from other causes is usually less well-developed, symptoms are less marked, and dropsy is uncommon.

Course and Prognosis.—In favorable cases the vomiting and headache soon stop, the fever gradually lessens and generally ceases in the course of a few days, the edema disappears more slowly, the urine, if scanty and discolored, becomes more abundant and of normal color, and the albumin, casts and blood-cells are finally no longer found. The duration of the attack is from 1 to 6 weeks, although a small amount of albumin and a few casts may continue for some months, being absent at some examinations and present at others. Complete recovery usually follows. The more serious cases may end fatally with uremic symptoms; such as vomiting, coma, or convulsions, with the urine greatly diminished or suppressed. The attack may occasionally last only a few days before death occurs in this way. In other instances death is the result of edema of the lungs or of the glottis, or of some complication such as pneumonia, meningitis, pleuritis, or endocarditis. Yet even in the severe cases recovery from the initial attack usually takes place. It is certain, however, that a very considerable number even of the milder cases pass eventually into the chronic form, although recovery may have seemed complete; manifestations of the chronic condition not appearing until months or even years have elapsed. This may indeed occur, as after scarlet fever, when the initial attack of nephritis was so mild as to have escaped notice. After acute nephritis of any sort anemia remains for a considerable time. The prognosis is influenced further by the age of the patient. The disease in infancy is liable to be fatal; after 3 or 4 years of age most cases recover from the initial attack. The nature of the cause is another prognostic factor of importance. The nephritis developing in the course of many of the infectious diseases, especially diphtheria, generally runs a favorable course. An exception exists in grippal nephritis which is liable to be hemorrhagic and more severe. Nephritis after scarlet fever recovers promptly in the large majority of cases, but has a distinctly greater tendency than some other forms to pass into a mildly chronic state, making the ultimate prognosis dubious in any case of post-scarlatinal nephritis. Certain symptoms are of unfavorable import. Thus the severity of the nephritis is in direct relation to the scantiness of the urine, the number of casts and red blood-cells, the retardation in the elimination of phenolsulphonephthalein indicating the degree of renal insufficiency, and the amount of dropsy, especially if this involves the serous cavities or the larynx. The amount of albumin present is a matter of less importance. Such uremic symptoms as obstinate vomiting, severe headache, dyspnea, stupor, coma, and convulsions, are always of serious import. Finally, the occurrence of complications naturally adds to the gravity of the prognosis.

Complications and Sequels.—Here are especially to be mentioned pneumonia, pleurisy, endocarditis, and pericarditis, as well as those depending upon the occurrence of edema of the glottis or the serous cavities. The sequel most to be dreaded is the development of chronic nephritis.

Diagnosis.—The distinction is especially to be made between a true acute nephritis and the forms of albuminuria previously described, including that dependent upon acute degeneration of the kidneys. It is certain that the amount of albumin in nephritis is usually larger than in the other conditions; but this alone is not sufficient to establish a diagnosis. So, too, the presence of casts is not in itself a criterion, since the occurrence of a small number of hyaline casts is frequent in the urine of healthy individuals, and a few granular casts and epithelial cells are observed in cases of acute degeneration of the kidneys. Consequently in acute infectious fevers without any general symptoms of nephritis, it may sometimes be impossible to determine from the urine whether the renal changes are truly inflammatory in nature. If, however, after the fever is over, small amounts of albumin and some granular casts, leucocytes and red blood-cells persist, the diagnosis of nephritis is justified. Dropsy of cardiac origin is distinguished by the evidence of valvular disease of the heart, and by the absence from the urine of the formed elements indicating inflammation of the kidneys. (See p. 174.) Edema in infants much more frequently depends upon a feebleness of circulation attending a marantic state. Should the case be one of nephritis the examination of the urine will reveal the presence of casts, although perhaps in small numbers.

Treatment.—**Prophylaxis.**—This is of importance because so often practicable. Children suffering from any of the infectious febrile disorders, and especially scarlet fever, should have the kidneys spared as much as possible. No drugs should be administered which are in any way irritating to these organs, and the elimination of the toxins should be favored and their evil effect lessened by increasing the urinary secretion; keeping the bowels open by saline laxatives; and making the skin freely active by warm sponging or of warm tub baths and by keeping the child warm. Elimination of toxins through the skin and the bowels must not be pushed to an extent which results in concentration of the urine. Confinement to bed is essential and the diet should consist of amylaceous foods and especially of milk, which is itself an excellent diuretic. In cases of scarlet fever these measures should be continued for at least 3 weeks, no matter how light the attack. The ingestion of water freely, either plain or alkaline, is of importance, and diuresis can be favored, if necessary, by the administration of citrate of potash. The influence of chilling of the surface of the body in the production of nephritis has been much discussed. A middle ground is probably the correct one; for although the danger may not exist when fever is present, it is admitted that congestion of the kidneys may result from exposure to cold; and it is reasonable to believe that nephritis may in like manner follow. Certainly in conditions of previous good health nephritis has followed surface chilling (p. 176), and it is a wise precaution, therefore, to avoid this as far as possible in the afebrile convalescent stage of acute infectious diseases.

The **treatment of the attack** itself varies with its severity. When the urine is but little altered and there is only a slight degree of edema or of the other symptoms, the treatment described for prophylaxis is to be continued. Acetate of potash is more successful than the citrate in some cases. Enemata of large amounts of warm normal saline solution may be of service in promoting diuresis. The action of the bowels should be kept free, with the precautions already mentioned. Rest in bed must last until all trace of albuminuria has disappeared, and the analysis should be made of the urine obtained at different times of the day. When the patient discontinues confinement to bed examinations should still be made in order to discover whether the character of the urine has been affected by this, and milk should still be the principal article of diet. Only in cases where the patient's general health is suffering from the long confinement to bed and the restricted diet is a cautious trial of a change in this particular admissible, meanwhile noting carefully the effect upon the urine.

In the severe cases the treatment must be more energetic. For widespread dropsy the employment of warm baths or hot packs followed by wrapping in blankets is of great value through the production of free perspiration. The hot-air bath or the vapor bath is often a more powerful agent. (Vol. I, p. 242.) Daily flushing of the colon with hot water is often serviceable. Dry cups or hot poultices frequently changed should be applied over the kidneys. Free purgation is also indicated. The importance of diet in this connection is great. The administration of a salt-free diet is sometimes followed by a rapid disappearance of dropsy. The action of milk is similar, perhaps on account of its small sodium-chloride content.

Measures similar to those recommended for dropsy are indicated for cases with great diminution or suppression of urine. Theocine or other caffeine derivatives are often useful diuretics in this condition, and digitalis is of aid also; but these drugs frequently disagree with the stomach. With the development of uremic symptoms with high arterial tension efforts must be made to increase the elimination of urine and reduce the tension by the procedures mentioned and by the employment of nitroglycerine. This remedy must be given in full dose frequently repeated. (See Vol. I, p. 226.) Uremic convulsions may sometimes be prevented or relieved by the combination of morphine and nitroglycerine given hypodermically. Chloral, also, in full doses should be given by the mouth or by enema, and lumbar puncture sometimes relieves promptly; but one of the most successful remedies in urgent uremia is bleeding by venesection or by leeching, followed by the intravenous injection of normal saline solution.

During convalescence from nephritis iron in some form is frequently needed for the treatment of the anemia which often remains. Basham's mixture is a very serviceable form. Should albumin and casts be slow in disappearing, removal to a warmer climate may be beneficial, together with the wearing of warmer clothing and the employment of food of an unirritating nature.

CHRONIC DIFFUSE NEPHRITIS

(Chronic Productive Nephritis; Chronic Interstitial Nephritis; Chronic Parenchymatous Nephritis; Chronic Desquamative Nephritis)

As in the acute form the lesions of the various tissues of the kidney are more or less combined, sometimes the degenerative changes predominating, sometimes the interstitial. No sharp distinctions of classification are consequently possible between the varieties, nor can the line of demarcation separating the acute and the chronic cases be sharply drawn; many of the former passing slowly into the latter. Two varieties are prominent, (1) that in which parenchymatous changes predominate (*chronic parenchymatous or degenerative nephritis*); (2) that in which the lesions are especially of an interstitial nature (*chronic interstitial* or *productive nephritis*).

Etiology.—Chronic nephritis is much less frequent in early life than is the acute form, yet probably is much commoner than many believe. Few cases are seen before later childhood. Yet the occurrence of congenital nephritis, even of the interstitial type, has been reported, as in instances published by Baginsky[1] and others. Sawyer[2] refers to the records of 13 cases under 10 years and of 8 between 10 and 15 years occurring in Great Britain. In a few instances the disease has been hereditary or familial, the tendency appearing even in childhood. Such cases have been reported by Brill and Libman,[3] Hellendall,[4] Heubner,[5] and Dickinson[6] (4 generations).

The causes of chronic diffuse nephritis are very diverse. Some cases are certainly primary, develop insidiously, and are essentially chronic from the beginning; although the majority of those apparently primary would doubtless be shown, if a complete history were obtainable, to owe their origin to some other disease from which the child had suffered possibly several years previously. Thus the earlier occurrence of one of the acute infectious disorders may give rise to the development later of the symptoms of chronic nephritis, even though no evidences of the acute form had been discovered. Of all diseases in childhood scarlet fever is that etiologically most prominent in this connection. Among other causes are the chronic digestive disturbances; long continued exposure to conditions of cold and dampness; diseases of the heart; hereditary syphilis; chronic tuberculous or suppurative processes; malaria; and rarely, in children, alcoholism. As already indicated attacks of acute nephritis may be followed by the chronic form; either immediately passing into this or apparently recovering, to be succeeded by evidences of the chronic condition after the lapse of several years.

Pathological Anatomy.—In characteristic cases of the *parenchymatous type* the lesions are chiefly in the epithelium, especially in that of the cortex. The cells are swollen and contain granular matter and sometimes fat-globules. They may be desquamated, and together with red blood-corpuscles, leucocytes, cellular detritus and casts, collect in and dilate many of the tubules. There is also a proliferation and desquamation of the cells of the glomeruli with distention of the capsules Growth of interstitial connective tissue in circumscribed patches takes place to a considerable extent in long-continued cases, compressing and causing atrophy of the tubules or the tufts. The macroscopic appearance of the kidney varies with the case. The renal capsule is more or less adherent and may tear away portions of the renal structure when it is stripped off. The kidney may be enlarged with a thickened white cortex, this change constituting the *large white kidney.* In other cases hemorrhage has taken place into the interstitial tissue, and the *large red kidney* is produced. In still other cases the kidney is mottled red and yellow, owing to the lack of similarity in the lesions of different portions, and in others it is not enlarged at all or is even smaller than normal, with a narrowed cortex.

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[1] Lehrb. d. Kinderkrankheiten, 1902, 992.

[2] Birmingham Med. Rev., 1903, LIII-LIV, 511.

[3] Mt. Sinai Hosp. Rep., 1901, II, 921.

[4] Arch. f. Kinderh., 1897, XXII, 61.

[5] Chronische Nephritis im Kindersalter, 1897, 35.

[6] Diseases of the Kidneys, 1877 378. Ref. Brill and Libman, *loc. cit.*

In the *chronic interstitial type* there is still greater over-growth of connective tissue. This may occur simultaneously with or may follow parenchymatous changes. The over-growth occurs in streaks or patches producing decided alteration of some portion of the kidney, with other areas between them but little altered in any way or showing parenchymatous changes. The new growth is at first chiefly cellular, but later is transformed into well-developed connective tissue situated along the course of the vessels and surrounding the tubules and tufts. Some of the tubules are compressed; others much dilated and even forming cysts; others destroyed. Casts of various sorts are visible in the organ. The glomeruli exhibit thickening of the capsules and of the capillary walls; or may be transformed into small fibrous knobs; or be completely atrophied and sometimes replaced by small cysts. The macroscopical appearance is that of the *granular kidney.* The organ is smaller than usual, with a nodular surface and with adherent capsule. It is firm on section and shows a narrow cortex which is mottled in appearance.

Although one form of lesion may predominate, both parenchymatous and interstitial changes are always combined in cases of chronic diffuse nephritis, and intermediate forms of all sorts occur. Even the lesions of acute nephritis may sometimes appear in a kidney which had previously suffered from a chronic inflammation. Some degree of amyloid change is a frequent attendant.

Symptoms.—As with the lesions it is convenient to discuss separately the symptoms of the two main types of the disease.

1. Chronic Parenchymatous Nephritis.—This is by far the more frequent form of chronic nephritis in early life. It may develop as a sequel to an acute nephritis either immediately or after a long interval, or the onset may be gradual without any such precedence being discoverable. In well-marked typical cases dropsy is a prominent symptom; either limited to the eyelids, ankles, hands, and external genitals; or widespread and involving the serous cavities as well (Figs. 317 and 318). The degree of dropsy varies from time to time, being sometimes nearly or entirely absent for a considerable period, and returning during exacerbations. With the dropsy is associated decided anemia, the hemoglobin and red blood-cells being much reduced, and the face often presenting a characteristic pallor. The urine contains albumin on most occasions when analysis is made, and generally in considerable amount, with numerous leucocytes, epithelial cells, and fatty, epithelial, hyaline, or granular casts. The daily secretion is much reduced and the specific gravity

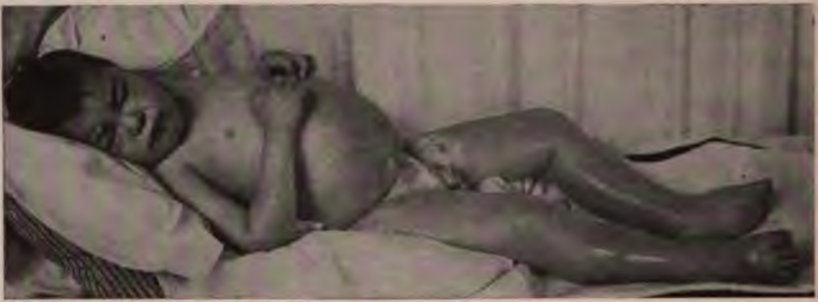

FIG. 317.—CHRONIC PARENCHYMATOUS NEPHRITIS CONSECUTIVE TO CARIES OF THE SPINE.

Patient a child of 6 years in the Children's Ward of the Hospital of the University of Pennsylvania. Exhibited wide-spread edema, yellowish-white tint to the skin, ascites, urine contained a small amount of albumin, with hyaline, light-granular and waxy casts. Death occurred suddenly without any decided previous improvement in the symptoms.

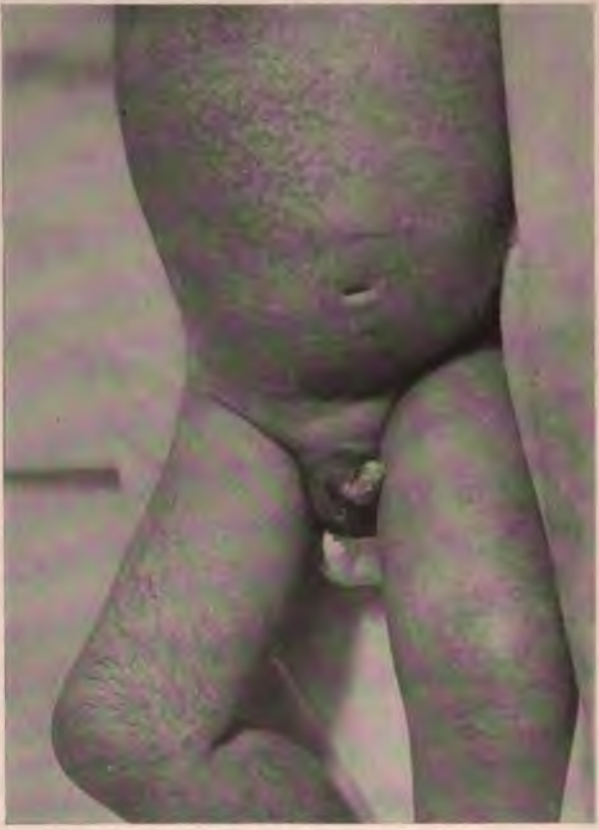

FIG. 318.—CHRONIC PARENCHYMATOUS NEPHRITIS.

Front view of same patient, as shown in the previous illustration. Exhibiting edema of the penis, and the white mottling of the abdomen produced by the stretching of the skin.

normal or high. During periods of improvement the amount of urine increases, the albumin grows less, and the casts diminish in number. Digestive disturbances may be present. Among these are loss of appetite, and occasional vomiting and diarrhea. There may be various nervous symptoms, such as irritability, loss of strength, headache, sleeplessness, dyspnea, and sometimes retinitis. As time passes hypertrophy and dilatation of the heart occur, especially of the left ventricle; but not to the extent observed in interstitial nephritis. Cardiac hypertrophy is to be considered evidence of increasing interstitial alteration of the kidney, as is increase of blood-pressure likewise to a certain extent; but Berkley and Lee[1] in studies upon 93 cases could not find any constant difference in the blood-pressure between the acute and the chronic cases.

The majority of the cases of chronic parenchymatous nephritis in childhood do not, however, exhibit the classical symptoms described, and the discovery that this disease is the cause of the symptoms observed is made only by the examination of the urine. Some such cases appear to be a prolongation of an acute nephritis of a mild type which developed during some infectious fever, and might be called subacute or latent in nature; the demarcation between them and acute nephritis on the one hand, and the prolonged chronic cases on the other, being not sharply marked. Among the principal symptoms shown in cases of this type are irritability, a moderate loss of strength, and a degree of anemia varying with the case. The child seems not quite well, yet without characteristic symptoms; edema is entirely absent or slight; and there is often headache or digestive disturbance. I have, for instance, seen the disease in this form manifested only by recurrent attacks of irritability with mild retinitis. As a rule, however, there are no ocular changes. The urine is little if at all diminished in amount, of normal specific gravity, and contains usually but a small quantity of albumin, or at times none, and a few granular, epithelial or oftener hyaline casts, perhaps covered by epithelial cells. There is no enlargement of the heart or increase of blood-pressure. The symptoms just described are those found during the exacerbations. In the intervals there are none whatever.

2. Chronic Interstitial Nephritis.—The characteristic form seen in adult life is of very unusual occurrence in children. The symptoms develop gradually, sometimes dating from infancy, the pathological condition being well established before any clinical evidences appear. The latter consist of anemia; emaciation; retardation of growth; darkening and harshness of the skin; digestive disorders; and various nervous symptoms, especially headache, insomnia, palpitation, restlessness, dyspnea suggesting cardiac asthma, and disturbances of sight, especially albuminuric retinitis. Heubner[2] reports well-marked albuminuric retinitis in a girl of 2½ years. The left ventricle is decidedly hypertrophied; the aortic second sound accentuated; the arterial tension high, and the arteries sometimes thickened and atheromatous. The urine is increased in amount and sometimes greatly so; pale; of low specific gravity; and contains only a small amount of albumin or none at all, and very few casts, chiefly hyaline. Uremic convulsions may occur as a terminal symptom.

Course and Prognosis.—The course of chronic nephritis is nearly always slow and the duration long. In the more favorable but well-characterized cases of the *parenchymatous type* the disease lasts usually several years with intervals of apparent complete health. Recovery may gradually take place, or oftener the lesions in the kidneys may slowly grow worse, the interstitial changes increasing and death finally ensuing, but perhaps not until adult life is reached. In the severer cases the prognosis is correspondingly worse and the course of the disease shorter, the most serious cases dying in a few months. Generally, however, the course is long, lasting several years. It is impossible to predict the duration of life in the individual case of parenchymatous nephritis, since the course is usually subject to many fluctuations, with times of improvement or apparent recovery followed by relapse. The fluctuations often occur without any discoverable reason, and independently of treatment. Death is the result of exhaustion, uremia, or some complication.

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[1] Amer. Jour. Dis. Child., 1917, XIII, 354.

[2] Kinderheilkunde, 1911, II, 502.

What has been said applies, however, particularly to the typical form of parenchymatous nephritis, and an exception exists in the cases described as subacute or latent, and which are almost peculiar to early life and constitute probably the majority of the instances of chronic nephritis at this period. The greater part of these will probably recover completely, while others at least become no worse, although the albuminuria continues. Heubner[1] reported comparatively complete recovery in at least 9 of 16 cases, the course of which had been following during several years. Nevertheless, the prognosis for the individual case is always uncertain, since interstitial changes may finally develop.

In the *chronic interstitial form* the actual course is even slower and more prolonged, although in many cases it may seem to be short because the disease has not been suspected until, after a long period, threatening uremic symptoms develop, or sometimes fatal hemorrhages occur, or other intercurrent complication causes death. The prognosis of this form is entirely unfavorable.

Among the unfavorable symptoms of chronic diffuse nephritis is the scanty secretion of urine continued in spite of treatment, this indicating decided renal insufficiency; or, on the other hand, urine of low specific gravity secreted constantly in large amount, the result of predominating interstitial changes. Widespread dropsy resisting treatment; great anemia; retardation of phenolsulphonephthalein excretion;[2] decided cardiac dilatation; and evidences of uremia are other unfavorable symptoms; and the development of complications adds to the gravity.

Complications.—Some of these are to be regarded as in part symptoms of the disease, among them being edema of the lungs, hydrothorax, ascites, and uremia. Among others are hemorrhage into the brain or elsewhere, pneumonia, pleurisy, pericarditis, endocarditis, and chronic valvular disease of the heart.

Diagnosis.—In well-marked parenchymatous cases the diagnosis rests upon the pallor, dropsy, and the characteristic alteration in the urine. The history of the attack, the longer course, and the absence of fever, distinguish it from *acute nephritis*, as does the greater tendency for blood to appear in the urine in the latter disease. The last is not, however, a safe criterion, since periods of a hemorrhagic condition may occur during the course of chronic nephritis. *Chronic passive congestion of the kidney* dependent upon cardiac disease is distinguished from nephritis by the fact that disease of the heart is present and by the coincident improvement in the renal and in the cardiac symptoms. There is often, however, a combination of chronic nephritis with chronic valvular disorder.

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[1] Ergebn. d. inn. Med. u. Kinderheilk., 1908, II, 609.

[2] For method of making the phenolsuphonephthalein test in children and its indications see Gittings and Mitchell, Amer. Journ. Dis. Child., 1917, XIV, 174.

Yet it is only the repeated systematic examination of the urine in all doubtful cases of disease of any sort which can lead to a recognition of many of the milder instances of chronic parenchymatous nephritis, and this is even more true of all cases of the interstitial form, since the symptoms here are usually so uncharacteristic. The albumin found in the urine in this way may, it is true, be the evidence of a *functional albuminuria*, but the diagnosis of the latter should not be too quickly made. The assumption should rather be that nephritis is present until careful study has proven otherwise. Since many cases of nephritis exhibit changes in the amount of albumin at different times even of the same day, these may readily be wrongly considered *orthostatic albuminuria*. The presence of granular and epithelial casts is the determining feature. (See Orthostatic Albuminuria, p. 164.)

Treatment.—The treatment consists in the relief of the symptoms during well-developed exacerbations, and in the prevention of their return. The management of the symptoms present, such, for instance, as dropsy, anemia and uremia, has already been described in considering acute nephritis. Reference may be made here to the claims advanced especially by Fischer[1] that the edema depends upon an acidosis, and that benefit follows the internal administration of sodium carbonate, combined with a normal or hypertonic sodium chloride solution given by rectal injection by the continuous drop-method, administering about 2 quarts in the first 24 hours. Further study is needed. I have seen mprovement follow in some instances, and the patient grow worse in others. In cases resistent to ordinary treatment decapsulation of the kidney as recommended by Edebohls[2] may be performed. This is often of great benefit, but the relief is liable to be temporary only. Cases of complete recovery are, however, on record.

The prevention of exacerbation is a matter of great difficulty and treatment in general is unsatisfactory. Dietetic and hygienic management occupy the first place. A diet largely of milk or one containing but little salt is indicated in some cases, when the appetite is not affected by the restricted regimen; yet it may be possible by judicious selection to give a varied diet free from salt over a long period with benefit. In the more frequent milder cases with recurrent development of the vague symptoms described (p. 185), a rigid diet is unnecessary except for a short time. Here vegetables and cereals may be given freely, and meat in moderate amount. Indeed, except during the exacerbations, this is true also of many of the more severe cases running a much prolonged course. Too long a continuance of a greatly restricted diet may do harm by undermining the general health. These remarks apply also to the matter of rest in bed, which may do harm if continued for too long a time. Moderate exercise, with rest for a portion of the day, is a better procedure, except during acute symptoms, with care that the patient never becomes over-fatigued. Change of residence is to be highly recommended to a warm, dry climate where life out of doors can be followed uninterruptedly. The clothing should be sufficiently warm to avoid all chilling. The frequent use of warm baths is beneficial, but cold baths and violent exercise are to be avoided. Tonic remedies are often needed, especially preparations of iron to combat the tendency to anemia.

[1] Œdema and Nephritis, 1915.

[2] New York Med. Rec., 1901, LX, 961.

AMYLOID KIDNEY

The cause of this condition is oftenest a long-continued suppurative process especially in the bones, but it may also occur as the result of tuberculous or syphilitic disorders in other parts of the body, and sometimes of chronic malnutrition from any source. It is much less common in early life than later. Of 146 cases collected by Fehr[1] only 6 occurred in the first 10 years of life. Post-mortem examination shows the kidneys firm, pale, and of a peculiar translucent, glassy aspect, and the section stains red when treated with iodine. The transformation usually begins in the vessels of the glomeruli, giving them a thickened waxy appearance, and spreads later to other vessels and to the walls and the epithelium of the tubules. Generally in well-developed cases the interstitial and parenchymatous changes of nephritis are present also to some extent, and conversely some degree of amyloid change may be found in cases of chronic nephritis. The amyloid degeneration always involves other organs as well as the kidney.

The **symptoms** are for the most part like those of chronic nephritis, but are commonly more or less lost sight of in those of the primary disease. Edema is frequent and the skin exhibits a peculiar, intense waxy pallor. Hypertrophy of the heart is uncommon; disturbances of digestion, especially diarrhea, are frequent; enlargement of the liver and spleen is discoverable. The urine is generally abundant, pale and contains albumin in large amount. Casts are usually few in number and are oftener waxy than in most cases of chronic nephritis.

The **prognosis** is unfavorable and death occurs, often after a prolonged course, from uremia or increasing debility, or is brought about by the illness to which the renal changes are secondary. The **diagnosis** rests upon the nature of the cause, the character of the urine, the coincident enlargement of the liver and spleen, and the absence in most instances of cardiac hypertrophy.

Treatment includes that directed to the primary cause and that appropriate for chronic nephritis.

TUBERCULOSIS OF THE KIDNEY

Etiology and Pathological Anatomy.—Except as one of the manifestations of general tuberculosis, involvement of the kidney by this disease is a comparatively uncommon affection in early life. It is nearly always secondary to tuberculosis elsewhere in the body, although rarely it may be the primary lesion and the cause of the clinical symptoms. The germs may enter the kidney in various ways, and the disease consequently occur in different forms. In the course of acute miliary tuberculosis it is a part of the general infection and the bacilli reach the organ through the blood. Small grey or yellow tubercles develop upon the surface and in the tissues of both kidneys, and even in the lining of the uriniferous tubules (*excretory* or *hematogenous tuberculosis*). This acute miliary form of the disorder is comparatively more common in early life than later, being seen oftenest in infancy and early childhood.

More frequently tuberculosis of the kidney is combined with tuberculous infection of other portions of the genito-urinary tract, as the pelvis of the kidney, the ureters, bladder, or other parts. In some cases it is secondary to tuberculosis in these other regions, the process being an *ascending* one. In much the greater number of cases the kidney is the first portion affected and may remain the only part; or the other regions may suffer secondarily to this, the process being a *descending* one. In this condition the kidney is perhaps involved directly from a tuberculous pleuritis or peritonitis, although this is rare; or the lesion may develop through metastasis from some other distant focus. This focus may be undiscoverable and the renal lesion then appears to be primary. Whether it is ever really primary would seem uncertain. Very frequently there is well-marked development of tuberculosis in other parts of the body.

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[1] Dissert. Bern, 1867. Ref., Senator, Nothnagel's Encyclop., Amer. Edit., 1905, 317.

The non-miliary, caseous form of renal tuberculosis is very much rarer in early life than later, and is oftenest seen in males. Hamill[1] was able to collect but 55 published cases, including 1 of his own, of what appeared to be primary renal tuberculosis in patients not over 14 years of age. Of these 32 were in boys and 14 in girls. The sex was not stated in the remainder. The lesions consist of cheesy masses of variable number and size which finally break down, leaving cavities. Interstitial inflammation and epithelial degeneration are found in the portion of the kidney adjacent to the caseous formation. The process is usually unilateral at the first, but finally the other kidney may become involved. The pyramids suffer more than the cortex, although sometimes the greater part of the kidney is destroyed. In other cases enlargement of the kidney may occur and the organ be distinctly nodular when the tuberculous masses are close to the capsule. Sometimes the inflammation spreads to the perinephritic tissue.

Symptoms.—Acute miliary tuberculosis of the kidney presents no special symptoms whatever. Those of the caseous form are indefinite, and are perhaps masked by the existence of the tuberculosis often present elsewhere in the body. There is usually fever, emaciation, debility, pain in the abdominal or renal region, and frequent and painful urination. The neighborhood of the kidney is tender to pressure, and sometimes distinct enlargement can be discovered if the disease has spread to the perinephritic tissues. Examination of the urine shows the presence of blood, pus, and tubercle bacilli, and sometimes shreds of renal tissue. The reaction is usually acid. Any albuminuria present depends upon the pus and blood. Casts are exceptional and due to a complicating nephritis.

Course and Prognosis.—The course of miliary tuberculosis is short, the patient soon dying from the general infection. The duration of caseous renal tuberculosis depends often upon the tuberculous condition elsewhere in the body. Occurring primarily it may last months or years. The final prognosis is unfavorable, death resulting from exhaustion or from extension of the disease to other parts of the body, unless operative treatment may have been successful.

Diagnosis.—This rests upon the known presence of tuberculosis elsewhere in the body; the symptoms described pointing to renal involvement; and the character of the urine. When the disease is apparently primary the distinction must be made from *pyelitis*. This is chiefly by the discovery in cases of renal tuberculosis of tubercle bacilli in the urine. *Renal calculus* produces the more typical pain or renal colic, little pus in the urine, and no fever unless an inflammatory condition is present as a complication. A Roentgen ray examination may reveal the stone. The existence of *tuberculosis of the bladder* can be determined by cystoscopic examination. Its presence is suggestive of involvement of the kidney also, and catheterization of the ureter will serve to determine whether this is the case. This method in combination with the phenolsulphonephthalein or other tests to determine the efficiency of the renal function (Gerahty and Rowntree)[1] will aid in settling the question as to whether only one of the kidneys is diseased. Often, however, the recognition of renal tuberculosis is difficult, and a positive diagnosis cannot be made without the discovery of the tubercle bacilli. Yet it must be remembered, that in not every case of this disease is the recognition of the microörganism in the urine possible; and likewise that other acid-fast germs, such as the smegma bacillus, must be carefully excluded in the examination. An inoculation of a guinea pig may be required.

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[1] Contributions to the William Pepper Laboratory, 1896

Treatment.—All possible means must be employed to sustain the general health; and measures used to control special symptoms as they arise. Operative treatment is to be adopted in suitable cases. It is appropriate when the disease appears primary, the condition of the general health is good, and but one kidney is involved and the other has been proven to be functionating entirely normally.

SUPPURATIVE NEPHRITIS

(Abscess of the Kidney)

Etiology.—Abscess of the kidney is uncommon in early life and is almost never primary. In most cases the cause is hematogenous in origin, through the occurrence of septic emboli of the renal vessels, with consequent infarction and the later production of suppuration. In other instances the inflammation may be an ascending process through the excretory channels; may reach the kidney by extension from neighboring suppurating tissues; or may result from the presence of a calculus or from trauma. No age is free and even the new born may exhibit the lesion.

Pathological Anatomy.—The lesions vary somewhat with the cause. In those of hematogenous origin both kidneys are usually involved and numerous small abscesses, or less numerous and larger ones, are present, the size depending to some extent upon the duration of the disease.

Symptoms.—In the variety resulting from septic emboli the symptoms are those of grave and increasing sepsis, and are due to the general infection of the patient. Only when with this condition is combined the presence of tenderness in the renal region, and of blood, pus, and tube-casts in the urine, is the diagnosis possible.

In other forms of abscess the symptoms are uncharacteristic and difficult of recognition because masked by those of the primary disease. This is especially true of abscess secondary to a pyelocystitis, since the urine is practically identical in appearance in the two conditions. The septic symptoms are, however, more marked after the renal tissue has become involved than when the pelvis alone is affected, and the amount of albumin is greater and is out of proportion to the number of pus-cells found. It may be possible, too, to discover by palpation a tender enlargement of the kidney. The examination of the urine will fail to aid in the diagnosis in those cases in which the abscess is walled off and the pus has not entered the excretory canals.

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[1] Journ. Amer. Med. Assoc., 1912, LVII, 811.

Course and Prognosis.—The prognosis of suppurative nephritis is very unfavorable in the metastatic cases, and the course usually short. In other forms the prognosis is also grave, but the case is not always fatal. It is best when there is free communication with the urinary tubules and ready discharge of pus. When this does not exist the pus makes its way in other directions, and the disease may then prove fatal, either rapidly or by long-continued suppuration and consequent exhaustion.

Treatment.—Treatment for metastatic abscess is valueless, and for the other form is that for the primary disease. In well-defined abscess operative interference may be of service. As in all such operations upon the kidneys the functional efficiency of the other kidney must be first determined.

RENAL CALCULUS

(Gravel)

Etiology.—The infarction of the tubules of the kidney with uric acid crystals, not infrequent in the new born, has already been referred to. (See Lithuria, p. 169.) Apart from this condition uric acid crystals often appear in the urine of infants and children of any age. These must not be considered as necessarily produced in the kidney. They may, it is true, be formed there, but as a matter of fact are oftener the result of changes in the urine after its evacuation. Although small calculi are not infrequent in the kidneys of infants, the occurrence of those of considerable size is rare. They have, however, been found in the new born. During childhood, especially before the age of 7 years and in males, calculi occur with comparative frequency. Civiale[1] found that of 5376 collected cases of calculous disease 45 per cent. were in children. There exists a certain undoubted endemic influence, since calculi are much more frequently reported from certain localities than others. An inherited tendency is also observed at times.

Pathological Anatomy.—The calculi oftenest consist of uric acid or its salts, although oxalates, phosphates, or other substances occasionally compose them. In over 500 calculi removed from children, according to Bókai,[2] 7⁄12 were composed of uric acid or urates and 4⁄12 of phosphate of lime. The stones may be many or few, and are of a size varying from that of gravel up to calculi which fill the entire pelvis of the kidney. Generally only one kidney is involved. Stones situated in the renal parenchyma produce a diffuse inflammation in the tissues surrounding them, and the kidney is often enlarged and has an uneven surface. When largely or entirely in the pelvis of the kidney, this is usually dilated and often a certain degree of pyelitis is present.

Symptoms.—In infancy the symptoms are indefinite or absent. Sometimes there are severe paroxysms of colic, renal in origin but supposed to be intestinal. After these attacks gravel of considerable size may be found in the urine. Characteristic symptoms are generally confined to older children. There is tenderness in the renal region and often the evidence of a moderate pyuria. Sometimes typical attacks of renal colic occur, due to the entrance of a stone from the pelvis into the ureter. These are productive of most intense pain in the region of the affected kidney, radiating to the lower part of the abdomen, the testicle, penis, and thigh. There is a constant desire to urinate, although but little urine is passed, and this sometimes contains blood. Vomiting may occur, and in some cases convulsions or collapse. The attack lasts a few hours or sometimes, with remissions, for several days. The symptoms cease as soon as the stone is expelled into the bladder, or if it returns from the ureter into the pelvis. Later the calculi which have reached the bladder are passed through the urethra, and are then found as sediment of the size of fine or coarser gravel, or as concretions even of the dimensions of a small bean.

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[1] Traité de l'affection calculeuse, Paris, 1838, 646.

[2] Traité des mal. de l'enf. (Grancher), 1904, II, 753.

Other cases have attacks of hematuria only without colic. These may be brought on by exercise or develop without directly exciting cause. This is true of calculi situated in the substance of the kidney. In many cases the urine in the interval shows evidences of the existence of pyelitis. Sometimes a stone becomes impacted in the ureter and produces a hydronephrosis (see p. 172) or pyonephrosis. In other instances the presence of a calculus in the kidney may be the cause of a renal abscess. It is to be remembered that patients sometimes pass a stone without any attack of colic having occurred.

Course and Prognosis.—The course is usually chronic. The calculus may exist a long time without any symptoms whatever; or there may be repeated attacks of colic at short or long intervals if the stone is not successfully passed on the first occasion. Even in this latter event there is a decided tendency to the formation of new calculi. The prognosis is, on the whole, good in children when the concretions are of small size; yet in long-continued cases death may result from anuria, uremia, pyelonephritis, or renal abscess.

Diagnosis.—This rests especially upon the attacks of pain as described, tenderness in one loin, the occurrence of blood in the urine, and the discovery at times of concretions, especially after an attack. The occurrence of pyelitis in combination with these symptoms is strongly corroborative. The employment of the Roentgen ray is of great value even in the case of small concretions.

Treatment.—The treatment should be preventive, directed especially to avoid recurrence of attacks due to the formation of new calculi in those subjects who have suffered previously. Water, preferably alkaline, should be taken regularly in large quantities; the digestion should be kept in good order to prevent the formation of the excess of urates which so often attends digestive disorders; and the diet should be unirritating and contain a minimum of flesh and a maximum of milk and vegetables. In the line of medicaments the administration of alkaline diuretics in sufficient amount to keep the urine alkaline is to be recommended. Various drugs have been employed as solvents but with uncertain results. During an attack of colic the patient should be immersed in a warm bath and opiates administered in sufficiently large dose, preferably hypodermically. A complicating pyelitis requires the treatment appropriate to this. In all frequently recurring cases with severe symptoms early operative interference is to be urged, before serious damage has been done to the kidney by the stone.

MORBID GROWTHS OF THE KIDNEY

Morbid growths of the kidney are of more frequent occurrence in early life than later, and are encountered especially in the first 5 years. The great majority are malignant in nature. In Aldibert's[1] collected series of operated cases, 3 were of a benign nature and 45 malignant. In Steffen's[1] series of 219 collected cases of growths of the kidney and adrenals in children, 34 were observed in the 1st year, 55 in the 2d, and 168, in all, in the first 5 years. Other statistics agree in placing the majority of cases at this early period. Thus of 130 cases of malignant growths collected by L. Taylor[2] 60 per cent. occurred in the first 3 years and over 80 per cent. in the first 4 years. The growths may even be congenital in time of appearance. In Walker's[3] series 12 of 138 collected cases of sarcoma had developed before birth. Probably a large number of renal tumors discovered later date their origin to antenatal life. There is little difference in the incidence in the sexes.

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[1] Rev. mens. des mal. de l'enf., 1893, XI, 501; 504.

Pathological Anatomy.—As a rule only one kidney is involved. The tumors are primary and malignant in the great majority of cases. One of the most frequent growths of this sort is sarcoma of different kinds, including mixed forms, embryonal adenosarcoma being the most frequent; while undoubted carcinoma is very uncommon. Renal hypernephroma, a frequent variety in which the tumor originates in misplaced adrenal tissue situated in the kidney, is sometimes malignant, sometimes benign. Malignant tumors may grow to a large size and fill a great part of the abdominal cavity, pressing upon and producing thrombosis in the vena cava or renal vessels, or causing hydronephrosis by compression of the ureter. Adhesions to all surrounding structures develop if the growth has been long present. Metastases to other organs may occur. The growth has its origin in the cortex or pelvis and may involve much of the kidney. Only exceptionally is it secondary through extension from a tumor of the suprarenal body or of the other kidney, or of other neighboring regions. The consistence of the growth varies. It may be firm, but is often quite soft and hemorrhagic and sometimes almost gelatinous. Cystic change may sometimes develop.

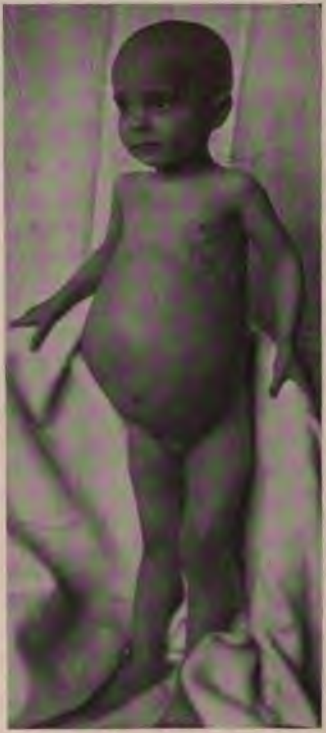

FIG. 319.—TUMOR OF THE KIDNEY.

Ida D., aged 2½ years. Had a swelling of the abdomen for some time, which had gradually grown larger. Entered the Children's Hospital of Philadelphia. Emaciation, diarrhea, no pain, leucocytes 9,000, hemoglobin 70 per cent. Large tumor of the right kidney, weighing 2½ pounds (1134) removed. Pronounced to be sarcoma. Operational recovery. Discharged. Further history unknown. Faint outline in the photograph shows the margin of the growth.

The benign tumors of the kidney, such as fibroma, lipoma, myoma and adenoma are of rare occurrence, usually small but sometimes of large size, and of slow growth.

Symptoms.—When first coming under observation the tumor is often already of considerable size, filling more or less of the abdominal cavity upon one side, and growing very rapidly (Fig. 319). It appears to project forward from the flank, and the anterior portion can be lifted distinctly by palpation with the hand in the lumbar region. It is more often soft than hard, and in some cases seems almost fluctuating. When firmer the surface may feel smooth or lobulated. The urine often contains blood and sometimes casts; but although this condition may be among the earliest symptoms, it is not a necessary one. Pain may be present especially in the renal region, but is usually absent. As the tumor grows debility, emaciation and anemia develop and progress, and digestive disturbances may be present, or other symptoms may arise depending upon interference with various organs by the pressure of the growth or upon the development of metastases. Among such symptoms are vomiting, diarrhea, edema, cough, and paralysis.

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[1] Die malig. Geschwulste im Kindersalter, 1905, 30.

[2] Amer. Jour. Med. Sci., 1887, XCIV, 461.

[3] Annals of Surgery, 1897, XXVI, 529.

Course and Prognosis.—The course is rapid, particularly in cases of sarcoma. In Walker's[1] series the average duration of unoperated cases of sarcoma from the time the symptoms were discovered, was 8.08 months, and in operated cases 16.77 months. Hypernephroma often runs a longer course. The prognosis of renal tumor is very unfavorable. Operation is sometimes immediately fatal from shock; sometimes, as stated, prolongs life by a few months, to terminate in recurrence; sometimes ends in recovery. In Steffen's list of 88 operated cases of tumor of the kidney and suprarenal body, 18 recovered completely.

Diagnosis.—This rests upon the presence of cachexia and hematuria, but especially, and sometimes solely, upon the discovery of the tumor and the determination that this is of renal origin. The decision is often of the greatest difficulty. Inasmuch as the great majority of cases of abdominal tumor in early childhood are renal or suprarenal, the presumption is always in favor of the diagnosis of one of these conditions. The fact that the growth appears to be largest in the renal region and projects forward also indicates its origin. Bimanual palpation is an aid in determining this. Further characteristics are the tympanitic percussion note produced by the passing of the colon, distended by gas, over the anterior surface of the tumor, and the relative movability of the growth as compared with sarcoma of the retroperitoneal glands, which, too, occupies a more central position. The distinction between renal and suprarenal growths is difficult and often impossible. Tumors of the kidney are oftener malignant and exhibit metastases, and are without the peculiar influence upon growth and nutrition which the suprarenal hypernephromata often exhibit. Splenic enlargements seem to take their origin from beneath the costal border in front; have a sharp edge, and often a marked splenic notch; and move more or less with respiration, while bimanual palpation from the renal region may show that it is not the kidney which is involved. Perinephritic abscess exhibits the fever characteristic of a purulent accumulation. A tumor arising in the liver is found by percussion and palpation to be directly continuous with it, and moves with respiration. Large fecal accumulations do not often extend to the flanks; and generally disappear after thorough purgation. Tuberculous masses in the loin may greatly simulate renal growths, but the other attendant symptoms and the history of the case aid in their recognition.

Treatment.—Nephrectomy is the only possible treatment open to us, and the earlier this is performed the greater the chance of recovery, both immediate and ultimate.

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[1] *Loc. cit.*

PERINEPHRITIS

(Perinephritic Abscess)

This consists of an inflammation in the fatty or connective tissue which together form the external capsule of the kidney.

Etiology.—It is not a frequent disease in early life, although Gibney[1] reported 28 personal cases of a primary nature occurring in children. In a series of 166 cases, Nieden[2] found but 26 in children. Less often the disease may be secondary to such causes as suppurative processes in the kidney; trauma; disease of the suprarenal bodies; exposure to cold; disease of the spinal column; pleurisy; septicemia; and the infectious fevers. In children it is oftenest primary, and the cause undiscoverable.

Pathological Anatomy.—The inflammation is usually unilateral, one side being affected about as often as the other. It starts in the connective tissue surrounding the kidney (*perinephritis*), or in the fatty tissue (*paranephritis*), and advances to suppuration in the majority of instances. The pus accumulates chiefly below the kidney and may extend in different directions and rupture in regions varying with the case; as the peritoneum, intestines, kidney, bladder, and pleura, but oftenest externally in the iliocostal space or above Poupart's ligament.

Symptoms.—In the acute cases the onset is marked by chilliness, high fever, severe constitutional disturbances, and frequently disorders of digestion. In those of slower onset the fever is moderate and all the symptoms develop more gradually and with less intensity. The most characteristic symptom is pain in the lumbar region on the affected side, or referred to the groin, hip, thigh, or knee. Any movement of the spine or the act of walking causes pain, and the patient progresses with the body bent toward the side involved. Physical examination of the lumbar region gives at first tenderness on palpation, later dullness on percussion and increased resistance, and finally an indefinitely outlined tumor, not moving with respiration. When of large size it may suggest fluctuation on palpation, while edema and redness may appear on the cutaneous surfaces.

Suffering increases as the disease advances, and the patient is necessarily confined to bed. Here the thigh is kept flexed upon the abdomen, and extension causes pain. Fever continues to be of an intermittent or remittent type. The urine is not often altered in character, and there are no urinary symptoms unless the kidney or the bladder is involved in the process, or interfered with by pressure.

Course and Prognosis.—The duration in acute cases is usually several weeks; in chronic cases sometimes some months; or even years if the cause persists and the pus has found an exit. The fever, pain and lameness often last some weeks before swelling can be discovered. The probability of complete recovery varies with the cause. The primary cases generally do well if properly treated, and recovery is fairly rapid after the pus has been discharged. All of Gibney's 28 cases recovered. Without operative interference the abscess finally ruptures in some of the directions mentioned. The prognosis when the abscess is secondary is influenced largely by the nature of the primary disease, which often overshadows the perinephritis in importance. The prognosis in unoperated cases depends upon the direction in which the pus has burrowed.

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[1] Chicago Med. Journ., 1880, XL, 561.

[2] Deut. Arch. f. klin. Med., 1878, XXII, 451.

Diagnosis.—This is often difficult at first, particularly in the cases developing insidiously and running a chronic course. The condition is especially to be distinguished from *disease of the hip-joint*. The latter is more chronic in onset; the lameness and deformity are much slower in developing; there is tenderness over the hip-joint; and passive movement here is restricted in all directions instead of in those involving extension only. *Caries of the spine* produces symptoms resembling those of perinephritis in many respects; but there is found the tender prominence of some of the vertebræ. After swelling in the renal region develops the diagnosis is much easier, although the possibility of the abscess being dependent upon spinal caries is not to be forgotten. Perinephritic abscess which ruptures into the pleural cavity is usually diagnosed as empyema.

Treatment.—Rest in bed is important. Early in the attack dry cupping or the application of an ice-bag or of hot poultices over the renal region may be employed in the hope of favoring resolution. Just as soon as the presence of pus is proven, by an exploratory puncture if necessary, the abscess should be opened by incision and drained. Later treatment is purely symptomatic.

PYELITIS

(Pyelocystitis)

The condition is variously denominated cystitis, pyelitis, pyelonephritis, and pyelocystitis, indicating the various lesions and combinations of lesions which may exist in different individuals, and which are but different manifestations of the same infection. Although doubtless many cases arise through infection from the bladder and in combination with it, and symptoms dependent upon inflammation of this organ may be present, the majority exhibit few if any vesical manifestations, and it is more convenient to consider pyelitis as an entity and to study cystitis separately.

Etiology.—The disease develops much oftener in early life than formerly supposed, and is, in fact, of decidedly frequent occurrence. In 225 unselected sick children without special symptoms examined by Fromm,[1] a urinary infection was discovered in 4.4 per cent. It is observed twice as often in children under 2 years as it is after that age (Thomson).[2] It is uncommon under 3 months of age. The disease is much more frequent in females, especially in infants, but not to the degree which is often thought. In 224 collected cases of pyelitis under 2 years, as given by Thomson[3] the incidence was as follows:

TABLE 86.—SEX IN PYELITIS

Among 77 patients under 6 months, 28 (36.3 per cent.) were boys.
Among 162 patients under 12 months, 40 (24.6 per cent.) were boys.
Among 224 patients under 2 years, 49 (21.8 per cent.) were boys.

It may depend upon the presence of a renal calculus; surface-chilling of the body; malformations of the kidney, bladder or urethra; the infectious fevers, such as scarlet fever, typhoid fever, diphtheria, or grippe; the existence of septicemia; tuberculosis or suppurative processes in the kidney; renal tumors; inflammations of the bladder; and disturbances of digestion, chiefly those of a diarrheal character. In older children, especially in males, renal calculus is one of the more frequent causes. In infants some other cause than this is operative, generally a digestive disturbance; or the disease appears to be primary. Whatever the cause the active agent is the penetration into the pelvis of the kidney of bacteria of some sort. These may enter by way of the blood-vessels, but it is generally believed that in the majority of cases in females in early life there occurs an ascending infection from the lower urinary passages, either by way of the mucous membrane or of the lymphatics. The bladder may be first attacked; but often the infection here, if occurring at all, is of a temporary nature and may have disappeared by the time the pelvis becomes involved. The development of pyelitis in male infants may be perhaps due to the migration of germs from the intestines into the bladder, but much oftener by other routes. After an extensive review of the literature and as a result of his own investigations, R. M. Smith[1] concluded that the infection is always by way of the blood, the germs entering the blood through the lymphatics. It would seem probable that any of these methods of infection is possible: ascending from the urethra; by way of the blood; or by the anastomosing lymphatics of the intestines and the urinary organs. The nature of the germ varies. In the very large majority of instances that present is the colon bacillus, alone or in combination. Among other germs found are the staphylococcus, streptococcus, gonococcus, typhoid bacillus, bacillus pyocyaneus, bacillus lactis aërogenes, bacillus proteus, tubercle bacillus and diphtheria bacillus. In spite of the frequency of vulvovaginitis, infection of the pelvis of the kidney by the gonococcus is uncommon. Fowler[2] found that in 136 cases of bacterial infection of the genito-urinary tract in children 80 per cent. were by the colon bacillus alone, or less often in combination; and Jefferys[3] found this germ in 67 out of 121 cases (55.37 per cent.).

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[1] Centralbl. f. Kinderh., 1904, IX, 367.
[2] Intern. Cong. of Med., London, 1913.
[3] *Loc. cit.*

Pathological Anatomy.—Calculous pyelitis is usually unilateral, but the pyelitis of infancy generally affects both kidneys. In acute cases there is a catarrhal inflammation of the mucous membrane of the pelvis, with redness and swelling, and a secretion of mucus, pus, desquamated epithelial cells and red blood-corpuscles. In the more chronic cases the mucous membrane is thickened, finally throughout all its layers, and is covered with a thick secretion of muco-pus, and the kidney becomes more or less involved (*pyelonephritis*), with widening of the tubules, the inflammation even extending into the cortical layer. If a large amount of pus accumulates in the pelvis of the kidney the condition of *pyonephrosis* develops.

Symptoms.—In typical cases of the acute form the symptoms are characteristic. With a sudden rise of temperature, reaching 103° to 105° F. (39.4° to 40.5° C.), preceded by chilliness, and continuing steadily elevated or oftener remittent or fluctuating, there is dull pain in the renal region radiating to the abdomen; sometimes tenderness over the kidneys; frequent micturition; debility; and loss of appetite. Urinalysis shows in general an acid reaction, normal specific gravity, and the quantity of the secretion unaffected or often scanty. There is but a small amount of albumin. Microscopical examination reveals pus-cells, red blood-corpuscles, epithelial cells of different forms, and perhaps a few hyaline casts. Bacteria may be present in varying numbers. Epithelial and granular

[1] Amer. Jour. Dis. Child., 1916, XII, 235.

[2] Garrod, Batten and Thursfield, Diseases of Children, 1913, 640.

[3] Quart. Jour. of Med., 1910–11, IV, 267.

casts indicate involvement of the kidney also (*pyelonephritis*). The amount of pus varies from time to time in the same patient, depending upon the degree of infection and whether the discharge is an uninterrupted one. In some instances the urine is quite turbid; in other cases only minute flocculi can be seen by transmitted light. If the pus is retained the urine will be temporarily nearly or quite free from it, and the fever and other constitutional disturbances correspondingly more marked.

The symptoms detailed are best seen in cases depending upon the presence of a renal calculus. In much the largest number, however, especially in those of a primary nature and in infants, they are subject to great variation from the type, are uncharacteristic, and the disease easily escapes recognition unless careful examination of the urine be made. This is true also of the cases secondary to diarrheal disturbances or to some acute infectious fever still present. In all such the general symptoms usually exceed the local manifestations. The fever may be moderate, or exhibit at times irregular elevations followed by depressions to or below normal. Sometimes several days pass without fever, to be succeeded by return of pyrexia (Fig. 320). The general condition may be fair, or there may be decided debility, emaciation, anemia, loss of appetite, constipation, and sometimes vomiting; suggesting the presence

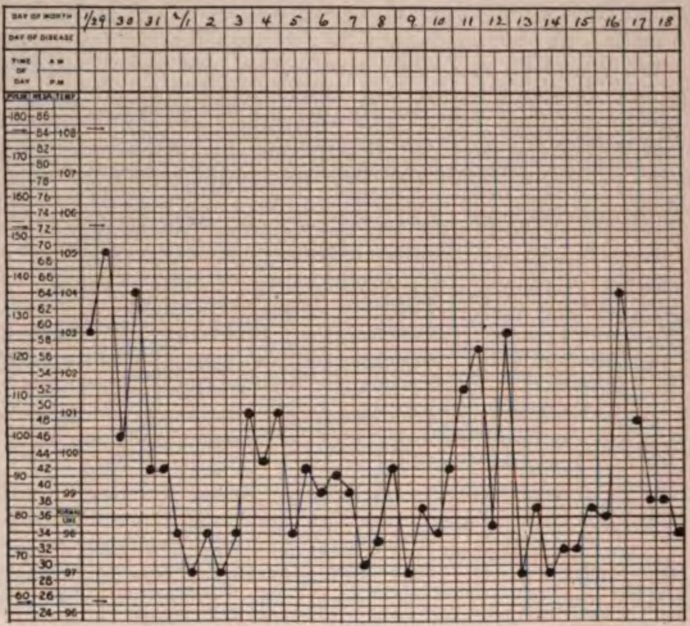

FIG. 320.—PYELITIS, TYPICALLY VAGUE SYMPTOMS USUALLY PRESENT, EXACERBATIONS OF FEVER.

Jeanette F., aged 2 years. Sudden onset Dec. 25, with vomiting and fever. Latter persisted, with fretfulness, malaise and cough. About Jan. 1, dysuria. Improved, but about Jan. 20 grew worse, with irregular fever, lost much weight, was anemic, restless, fretful, and very weak. Entered Children's Ward of the University Hospital, Philadelphia, Jan. 28. In hospital same symptoms continued, then gradually improved. Physical examination negative, except that urine was acid and contained pus, and on culture colon bacilli, and there was tenderness in the left renal region.

of an acute digestive disorder. Disturbances of micturition are oftener absent than present in uncomplicated cases; and this is true, too, of localized pain and tenderness. Cases of chronic pyelitis exhibit often merely pyuria; fever and other constitutional symptoms developing only during the acute exacerbations which are liable to occur.

Course and Prognosis.—The severity and duration are very variable. The disease is liable to be more severe and the mortality higher in infants under 2 years. The acute attack lasts from 2 to 4 weeks in the majority of cases properly treated; and still longer if unrecognized. In the acute less severe cases the fever may disappear in a week (Fig. 321). The symptoms are exceedingly liable to recur after an interval of weeks or months. This tendency to recurrence, or a general intractability sometimes seen, is likely to make the course long drawn out, and the condition may sometimes last for years without, however, in most instances doing the patient serious damage, except for the constant danger of a decided degree of complicating nephritis developing. Occasionally cases are very severe from the onset, and may run a rapidly fatal course, but these are uncommon and occur only when there is involvement of the renal tissue as well; while other severe instances of a chronic nature may gradually exhaust the patient. Sometimes in chronic cases the discharge of pus becomes obstructed and a pyonephrosis develops. In Thomson's[1] 71 cases the mortality was 9.8 per cent., limited entirely to patients under 2 years of age; none of the 23 cases over this age terminating fatally. This makes the mortality in those under 2 years 14.6 per cent. The mortality in 84 of Göppert's[2] 104 cases, most of them under 2 years of age, was 12 per cent. Under proper treatment, however, the prognosis of acute pyelitis is good in the large majority of cases. It is more uncertain and unfavorable when dependent upon some persisting acting cause, such as calculus, renal tuberculosis or other local factors.

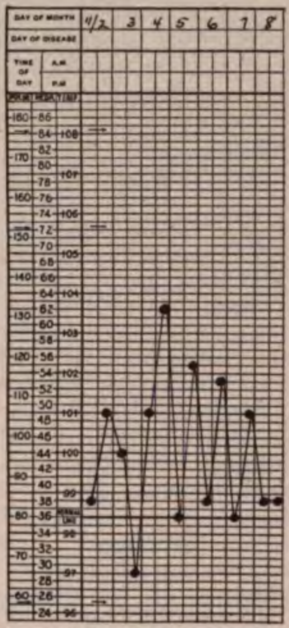

Fig. 321.—Acute Pyelitis with Short Febrile Course.

Jerry B., aged 7 years. Fever began Nov. 1. Admitted to Children's Ward of the Hospital of the University of Pennsylvania, Nov. 2. No special symptoms, did not appear ill except for the fever. This disappeared after a week, although pus was still present in the urine.

Complications.—The most frequent of these is probably cystitis, and some degree of involvement of the renal tissue (pyelonephritis). Also associated with pyelitis, either as cause or sequel, are perinephritis, pyonephrosis, hydronephrosis, malformations of the kidney, morbid growths of the kidney, suppurative nephritis, renal calculus, and tuberculosis of the kidney. In severe cases long-continued bronchopneumonia or some other intercurrent malady may be the cause of death.

Diagnosis.—This rests in typical cases upon the irregular fever, prostration, and other constitutional symptoms; pain and tenderness in the renal region; and the presence of pyuria with acid urine. In cases without subjective local manifestations, forming as they do the majority, it is only by the pyuria that the diagnosis can be made. All sorts of inflammatory diseases may be supposed to be present, and an incorrect diagnosis made unless an examination of the urine is carried out. Every infant or child with fever of obscure nature should be under suspicion of pyelitis and should have the urine studied carefully, not once but repeatedly. There is hardly another disease so readily overlooked, and in connection with which errors in diagnosis are more easily made. Yet the presence of pus in the urine may be dependent upon a number of causes and is not in itself an evidence of uncomplicated pyelitis. To justify a diagnosis of pyuria there should be at least 6 to 8 leucocytes in every field (Göppert), using uncentrifuged urine. It is to be remembered, too, that at the time of a decided rise of temperature no leucocytes may be present in the urine, the elevation being produced by the retention of the pus. Among the pus-producing conditions, cystitis is to be distinguished by the pain over and the evidences of irritability of the bladder. Although disturbance of micturition may occur in uncomplicated pyelitis as a reflex symptom, it is not frequent and not so marked as when the bladder is attacked. When the bladder is involved alone there is no pain or tenderness in the region of the kidney. Cystoscopic examination will aid in making the differential diagnosis. The rupture of a renal abscess into the pelvis of the kidney or the discharging of a pyonephrosis is the cause of a sudden appearance of a large amount of pus in the urine, at which time the symptoms of abscess ameliorate. Nephritis occurring alone gives rise to epithelial or granular casts and a few leucocytes, and if complicated by pyelitis, to a larger amount of albumin than can be accounted for by the presence of pus. Renal calculus produces the colic, hematuria and other symptoms characteristic of this disease. Tuberculosis of the kidney generally exhibits evidences of complicating tuberculosis elsewhere in the body, and tubercle bacilli may perhaps be discovered in the urine.

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[1] *Loc. cit.*

[2] Ergebn. d. inner. Med. u. Kinderh., 1908, II, 30.

As regards the bacteriological variety of pyelitis present, cultures from a catheterized specimen will reveal the nature of the germ. The agglutinative reaction may also be obtained with the blood in cases of infection with the colon bacillus. The urine is always acid in the instances of infection with this germ. This is not true of the rarer cases dependent upon streptococcic or staphylococcic infection (Langstein).[1]

Treatment.—In the way of prevention, the external genitals should be kept as clean as possible in the case of female infants, the movement of the nurse's hand in cleansing being from before backward. All irritating causes, such as gravel and calculus, must be sought for and appropriate treatment employed. Should it be necessary to use a catheter at any time in subjects of any age, the most scrupulous aseptic precautions must be taken. The giving of an abundance of water is of value, and when infants refuse this, as sometimes happens in severe cases, it may be necessary to administer it by the stomach-tube, nasal-tube, enteroclysis, or even subcutaneously. Inasmuch as the colon bacillus is the cause in the great majority of cases of pyelitis, and as this requires that the urine be acid for its proper growth, the rendering of it alkaline by the administration of alkaline salts has been used with great success as a curative measure. The amount required is often considerable, and can be determined only by testing the urine. The ingestion of 5 grains

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[1] Pfaundler and Schlossmann, Handb. d. Kinderh., 1910, IV, 101.

(0.32) of potassium citrate with sodium bicarbonate in even larger amount 3 to 5 times a day will probably be necessary for an infant of a year, and often the quantity must be 3 or 4 times this. In cases in which the urine is already alkaline and the colon bacillus has not been found, benzoic acid may be given. One of the favorite remedies is hexamethylenamine, but this is best suited to cases in which the reaction of the urine is acid. Infants of a year require from 1 to 3 grains (0.065 to 0.194) several times daily. Salol is also used successfully in doses of ½ grain (0 032) 5 to 8 times a day under 1 year of age. My own experience is decidedly in favor of the alkaline treatment, and it is to be borne in mind that hexamethylenamine in full dosage is capable of producing hematuria. Treatment with hexamethylenamine and with alkalies cannot well be combined as the former requires an acid urine for its action.

Extensive employment has been made of autogenous vaccines in the treatment of pyelitis, with varying conclusions. There is reason to believe that they are of service in some instances, and they should be tried in obstinate cases. In addition to these measures the patient should be kept in bed while fever is present, and an unirritating diet employed. Complications such as pyonephrosis and renal calculus may require surgical interference.

CHAPTER III

DISEASES OF THE BLADDER

MALFORMATIONS

A very few instances of **absence of the bladder** have been reported, and the occurrence of the rudimentary presence of **two or more bladders** is equally uncommon. **Congenital diverticula** have also been observed, and sometimes a complete or partial obstruction in the course of the urethra produces an enormous **congenital dilatation.** An instance of this is shown in the illustration (Fig. 322).

Persistent urachus (*umbilical fistula*) is a rare malformation in which the urachus remains patulous and the urine may be discharged through the navel. It may be associated with congenital occlusion or stenosis of the urethra. The condition has been studied especially by Herrmann.[1]

Exstrophy of the Bladder.—This is the only malformation of clinical importance; an uncommon condition seen with few exceptions only in boys. There is a defect in the anterior wall of the bladder itself and of the abdominal parieties over it. In the complete cases there is fissure of the pubic bone and of the penis, or the clitoris, and other anomalies of development are often combined. The posterior wall of the bladder is visible as a red, roughened area in which the orifices of the ureters can be seen. The adjacent cutaneous surfaces are excoriated by the urine, and the offensive ammoniacal odor of decomposing urine is constantly present. The penis is small, and procreation is not possible. In females the vagina may be absent or be replaced by a cloaca including vagina and rectum. The chief danger is the development of a pyelonephritis, which proves fatal to the majority of sufferers from exstrophy before adult life is reached.

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[1] Inaug. Dissert. Berl., 1885. Ref., Baginsky, Lehrb. d. Kinderkr., 1902, 1020.

The condition can be dealt with only surgically, and even in this way not satisfactorily in the majority of instances. A plastic operation may be attempted to replace the anterior wall of the bladder as far as possible. In other cases transplantation of the ureters into the sigmoid flexure or the rectum has been performed, and the patient has been able to retain urine in the bowel fairly well.

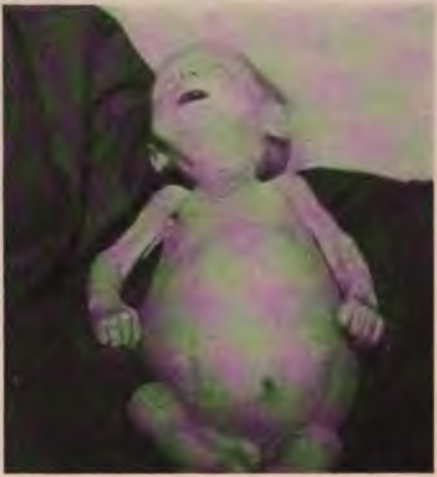

Fig. 322.—Congenital Dilatation of the Bladder.

Child, aged 3 months, with partial hypospadias. The abdominal distention was noted at birth, and steadily increased. The autopsy revealed a distended bladder extending to the diaphragm, and containing over a quart (946 c.c.) of urine. *Courtesy of Dr. J. Madison Taylor.*

PROLAPSE OF THE BLADDER

This is an uncommon condition, either congenital or acquired, occasionally seen in female children. A portion, or occasionally, all, of the wall of the bladder projects through the urethra or even through the vulva, sometimes as the result of trauma or of the straining accompanying dysentery or cystitis. The mucous membrane of the bladder appears as a deep-red or bluish-red mass which becomes smaller under pressure. Treatment consists in the reduction of the prolapse, and the prevention of recurrence by suitable compresses and bandages, or by a plastic operation upon the dilated urethra should this be necessary.

VESICAL CALCULUS

Etiology and Pathological Anatomy.—Although stone in the bladder is not an uncommon affection in children, occurring nearly or quite as often as in adult life, the incidence seems to vary with the locality. It is less frequent in the United States than in many other countries, such, for example, as England, India, Russia, and Hungary. During childhood it is observed especially often between the ages of 2 and 7 years, according to Bókai's statistics,[1] yet it may occur even in infancy.

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[1] Gerhardt's Handb. d. Kinderkr., IV, 3, 580. Jahrb. f. Kinderh., 1895, XL, 32.

Of 1621 cases of calculus in children studied by Bókai[1] 163 (10 per cent.) were in the first 2 years of life. Boys suffer from vesical calculus much more frequently than do girls, owing to the fact that the short and wide female urethra allows the stone to be discharged while still small and undiscovered. The calculus usually begins to be formed in the kidney, and increases in size in the bladder by repeated deposits upon the original nucleus, its final dimensions varying with its age and composition. As in the case of renal calculus it is oftenest composed of urates, but if the urine is alkaline the salts deposited in the bladder may be phosphates. Sometimes a foreign body introduced into the bladder forms a nucleus for a stone.

Symptoms.—Among early symptoms is vesical irritability or enuresis. There are often also attacks of pain during micturition, especially toward its close. This is felt in the neck of the bladder or the end of the penis or in the vagina, rectum or perineum. Tenesmus is frequent both on urination and defecation, and may produce prolapse of the rectum. Often there is a sudden arrest of the discharge during micturition, accompanied by severe pain and straining. Priapism is of common occurrence, and incontinence may become persistent, the urine constantly dropping from the penis, decomposing, and irritating the neighboring skin. As inflammation of the bladder develops and increases there is a proportionate increase in the severity of the symptoms.

The urine exhibits either no change at all, or in cases with actual cystitis a small amount of mucus and pus and occasionally red blood-cells in small numbers. If the stone is of large size bimanual palpation with one finger in the rectum may possibly reveal its presence; but the only certain diagnosis is cystoscopic or by exploration with a sound or examination with the Roentgen ray. This last is not so serviceable as in renal calculus, since the stone is often composed of uric acid which is not revealed by the ray. If the disease is severe and long continued, the **prognosis** is uncertain on account of the exhaustion which is produced. The only **treatment** is surgical.

FOREIGN BODIES IN THE BLADDER

Occasionally boys and girls, especially at the age of puberty, introduce foreign bodies into the urethra, and these may pass into the bladder. The nature of these is varied, among them being hair-pins, pencils, shot, beads, etc. In the bladder they may give rise to inflammation or form the basis for a calculus, with the symptoms of these diseases. Treatment consists in the removal of the body by surgical procedure.

CYSTITIS

Etiology.—This affection, occurring alone with well-marked symptoms, is not common in older children. In infancy and early childhood it is oftener encountered in combination with pyelitis and adding to the severity of this; but even at this age it is not of frequent occurrence, so far as clinical manifestations are concerned. The very large majority of cases of pyelitis exhibit no symptoms referable to the bladder (Göppert,[2] 3 in 104 cases). There is a great preponderance of cases in girls. Among the causes are vesical calculi, foreign bodies, tumors, the rupture of an abscess into the bladder, an ascending spread of the gonococcus or, much oftener, the colon bacillus, or the action of other germs transmitted by way of the blood. It is met with sometimes in the course of infectious diseases, especially typhoid fever; or may develop as a complication of intestinal disorders, oftenest in infancy. Chilling of the body appears to be a predisposing cause in some instances. Tuberculous cystitis is a rare local manifestation of tuberculosis in children, associated with evidences of tuberculosis elsewhere in the body (see p. 211). By far the most common cause is the colon bacillus, and on this account the condition has often been denominated *colicystitis*.

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[1] *Loc. cit.*

[2] Ergebn. d. inn. Med. u. Kinderheilk., 1908, II, 30.

Pathological Anatomy.—In the acute condition the mucous membrane is redder than normal, swollen, infiltrated, exhibits hemorrhagic streaks, and is covered with mucus or muco-pus. In the more chronic cases the mucous membrane is much thickened, possesses very prominent rugæ; is grey in patches and hemorrhagic in others, and may show superficial or deeper ulcerations.

Symptoms.—The constitutional symptoms include fever, malaise, fretfulness, restlessness, and loss of appetite; but except in the severe cases are less marked than in pyelitis. The local disturbance varies considerably in intensity. It is usually greater in older children, the symptoms then consisting of pain and tenderness in the region of the bladder, dysuria which may be intense, and a constant desire to urinate although but little urine is passed at a time and that only with painful straining. In the more chronic cases the severe frequent straining may produce prolapse of the rectum, and there may be constant dribbling of urine. In the milder instances, especially in infancy, the only local symptom, apart from the character of the urine, is perhaps the occurrence of crying before urination. The urine in the acute cases is cloudy in appearance; usually acid, although often quickly becoming alkaline after it is evacuated; and contains pus cells, mucus, vesical epithelium, bacteria, a varying number of red blood-corpuscles, and a small amount of albumin. In the more chronic cases pus cells and mucus are present in larger amount and the reaction is often alkaline from decomposition within the bladder, and in this event crystals of triple phosphate and urate of ammonia are found.

Course and Prognosis.—The duration is very indefinite, depending upon the cause and the possibility of the removal of this. Even in favorable cases under treatment the disease is prolonged and relapse readily occurs, as in the case of pyelitis. If there is a definite, removable cause, and the attack is acute, recovery should follow. The prognosis of cases depending upon the colon bacillus is decidedly better than when other germs are operative. The longer the chronic cases have continued the greater is the severity of the lesion and the intractability of the symptoms. There is always danger of the extension of the inflammation to the kidney with a serious result following.

Diagnosis.—As a rule this is easy. Only to be distinguished are those conditions which may produce reflex symptoms connected with the bladder, but a study of the urine will exclude these. The characteristics of cystitis are the frequent painful urination and the alteration of the urine. The combination of a small amount of albumin, and an absence of casts, with the presence of a rather large quantity of mucus and pus excludes nephritis. Decided severity of vesical symptoms indicates cystitis rather than pyelitis. To make a positive diagnosis, however, between these two a cystoscopic examination may be necessary. If cystitis is found in this way, it may be possible to catheterize the ureters in order to discover whether the urine obtained indicates that pyelitis is also present.

Treatment.—The cause should be sought for and removed if possible. In the acute cases rest in bed is needed, with an unirritating diet and large amounts of water. In these and in other matters, such as the administration of alkalies when the urine is acid, hexamethylenamine, or salol, the treatment is much as for pyelitis (p. 200). Benzoic acid should be used if the urine is alkaline. In addition it is necessary to relieve pain and straining by the employment of hot or cold applications over the bladder, or the administration of opiates if required. In severe acute cases not yeilding after such methods of treatment, and in many chronic cases, lavage of the bladder is indicated, employing a dilute sterilized boric acid solution (2 to 3 per cent.), and later weak solutions of permanganate of potash (1 : 8000), bichloride of mercury (1 : 10,000), nitrate of silver (1 : 6000), argyrol, or silvol (5 per cent.). The stronger injections should be left in the bladder for a short time and then washed out with sterile water.

SPASM OF THE BLADDER

(Vesical Irritability)

Etiology.—In some form spasm of the bladder is of common occurrence in children, although it is more frequent in early childhood than later. Among the numerous causes are the sudden chilling of the surface of the body; excitability of the nervous system; vesical calculus; and the secretion of a highly concentrated urine, as from febrile disease, indigestion, or other source. It may also be a reflex symptom of renal calculus or of lesions of the rectum, vulva, urethra, hip-joint, appendix, or other neighboring structures.

Symptoms.—These vary greatly in intensity, and to some extent in character. In the less frequent form there occurs a spasm of the sphincter, making the child unable to excrete the urine present. More commonly the detrusor apparatus is in an irritated condition, resulting in very frequent emptying of the bladder of small amounts of urine. The former condition is attended by severe pain and straining efforts without the passage of urine during the attack. The latter exhibits symptoms similar to these, but with the passing of urine very frequently, and sometimes only in drops in the severer cases. In milder instances of this type there may be only frequent desire to urinate with no actual pain and no straining. Spasm of the bladder exhibits no alteration in the character of the urine other than the concentration which is so frequently an exciting cause. When dependent upon this or upon some temporary irritability of the bladder the attack is generally of short duration. If produced by a reflex disturbance from disease in another organ, or due to a vesical calculus, the course of the disease is variable, and there is often a tendency to recurrence of the attacks.

Treatment.—This consists in seeking carefully for the cause, and treating this as far as possible. When the disease is produced by the acid character of the urine or simple nervous irritability, alkaline diuretics should be given in full doses, water ingested freely, and belladonna or the bromides administered. A useful remedy during the attack of spasm of the sphincter is the application of hot compresses over the bladder or the giving of a warm tub-bath. If this is not successful catheterization may be necessary

ENURESIS

(Incontinence of Urine)

Incontinence of urine may be due to organic or other causes which render the retention of urine impossible. This has already been referred to under Incontinence of Urine (p. 164). Here only will be discussed the lack of control which is dependent, at least in part, upon a neurosis. and which could with equal propriety be considered under Diseases of the Nervous System. It is one of the frequent disorders of childhood. In 1657 children in my service in the Out-patient Department of the Hospital of the University of Pennsylvania, as reported by Ostheimer and Levi,[1] 85 (5.13 per cent.) were suffering from this disease.

Etiology.—The influence of age is to be considered in establishing a definition of this disease. The time at which an infant should learn control of micturition varies greatly with the individual and with the care which has been given to training. Some exceptionally well-taught infants acquire the control during the day-time even within the first 6 months of life; but as a rule the 1st year will be passed before it is gained. By the end of the 2d year every normal healthy child should have learned while awake to restrain the desire to urinate except into a vessel, and lapses during sleep should not frequently occur. If after the age of 2 years there is frequent bed-wetting, enuresis may be said to exist. The age, therefore, at which the disease can begin is that of 2 years, and it remains frequent until near the time of puberty. Sex exerts but little influence. Many cases show distinctly the effect of heredity, the parents suffering from nervous disturbances, or enuresis having been present during their childhood, or existing in the brothers or sisters of the patient. Most of the cases are a persistence of the condition normal to infancy, which period is characterized by a powerful detrusor apparatus and an insufficient sphincter control of the bladder. A smaller number develop it after the normal control had been acquired.

In considering other causes it is important to remember the method by which the disease may be brought about. Urination is a reflex act in which are concerned the wall of the bladder, the efferent and afferent nerves connecting this with the spinal centers, and these centers themselves. There exists, too, the possibility of reflex irritation of this apparatus by disturbance arising in other parts of the body, generally closely associated or not far distant, but sometimes entirely remote. Finally there is the inhibition exerted by the brain, which controls the act of urination even during sleep. Disorders in any part of this apparatus are capable of producing enuresis, but some of them are of little importance. Very prominent, varied, and influential among the causes is reflex disturbance outside of the direct reflex arc. In this class are preputial adhesions or phimosis with attendant irritation, such as balanitis; a narrow meatus; vulvovaginitis; adherent clitoris; the local irritation produced by masturbation; renal calculus; irritability of the rectum of various sorts, such as fissures, thread worms, inflammation, or even the presence of a mass of feces in the rectum during the night; irritation of other portions of the gastrointestinal canal; adenoids in the nasopharynx; foreign bodies in the nose, etc. These agencies may reflexly stimulate the detrusor urinæ to contract or the sphincter vesicæ to relax. Another class of causes of reflex irritation arises in the bladder. Here are to be mentioned vesical calculus; cystitis; and especially irritation of the mucous membrane of the organ by undue acidity of the urine, or by an excessive secretion and consequent distention of the bladder by urine of low specific gravity.

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[1] Journ. Amer. Med. Assoc., 1904, Dec. 17.

Yet no reflex cause within or without the bladder is sufficient alone to produce enuresis, and a combination with some other etiological factor is necessary. Among such factors may be some intrinsic disorder of the bladder itself, there existing perhaps an undue excitability of the muscular walls or a pathological weakness of the sphincter. Another group of causes consists in disturbances of the nerves connecting the bladder with the spinal centres, and of the centres themselves. Any agent producing nervous excitability or general debility of the child is to be placed in this category. Among such are acute febrile diseases; malnutrition; anemia; chorea; epilepsy; headache; neurasthenia; hysteria; masturbation; improper food or clothing; fright or other intense excitement; and various other conditions, not forgetting the influence of habit, and possibly that of thyroid insufficiency.

In addition to all these various etiological factors there remains to be considered the control of the brain, which, in the majority of cases, is sufficient in the day-time to prevent the evacuation of the bladder. During sleep the complete removal of this control may be favored by a number of causes which make the sleep unusually profound, such as ingestion of a hearty evening meal; the sleeping upon the back; fatigue; or the use of too heavy bed-covers.

Symptoms.—In *enuresis nocturna* the urine is passed into the bed only during sleep. Patients with *enuresis diurna* are unable to control the bladder during the waking hours. This latter is much less frequent occurring alone, but is often combined to a varying degree with the nocturnal form. In the series of Ostheimer and Levi[1] 53 were nocturnal only; 35 both noctural and diurnal, and only 2 purely diurnal. The mildest cases of nocturnal enuresis wet the bed only occasionally. From these there are all grades up to the inveterate bed-wetters, where scarcely a night passes without the accident occurring from one to several times. Sometimes children, especially older ones, dream that they are passing urine into a vessel or the toilet, and may then perhaps be awakened by the accident; but as a rule the child is not roused by the involuntary micturition. There is no fixed time in the night when the bed-wetting occurs. Perhaps oftenest it is during the first sound sleep before midnight; in others it is toward morning; in others at variable hours.

Diurnal enuresis often develops after nocturnal enuresis has continued for some time. In this form there is a too frequent desire to evacuate the bladder, perhaps coming on so suddenly that the clothing is wet before the child can retire. In bad cases the patient is constantly wet with offensive decomposing urine. In some children play, exercise or sudden laughing, or fright or other emotion is followed immediately by evacuation of the bladder.

The condition of the general health varies with the case. Often the patient is of a decidedly neurotic temperament or exhibits some evidence of malnutrition; in other cases some other nervous condition is associated; in still others—and these, I think, the majority—the general health appears to be excellent. Not infrequently the psychic state is affected by the mortification which the disease produces or by the punishment which has been too freely administered.

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[1] *Loc. cit.*

Course and Prognosis.—The duration depends largely upon the cause. If following some acute illness the disease lasts usually but a short time. If it is the persistence of the infantile condition, the course is long; and the longer it has lasted the more resistant is the affection to treatment. The disorder is seldom absolutely continuous. Periods of days, weeks or months may occur when there is little if any incontinence. This is more liable to happen in the summer-season than in the winter. The disease may occasionally appear to be cured and then redevelop with the establishment of puberty. This is, however, not common.

In general the prognosis is good. The majority of cases which come under treatment in early childhood can be cured, although it may require months to accomplish this result. Those first seen in later childhood are more difficult to relieve. Many cases, however, which at first seem not severe are found on trial to be most intractable to treatment. Fortunately the disorder usually disappears of itself as puberty is approached, and may, indeed, cease at any time before this without treatment and often without discoverable reason. Occasionally in girls it does not disappear at puberty, and then offers a very unfavorable prognosis.

Diagnosis.—Cases of incontinence of urine due to malformations or to paralysis of the bladder suffer from dribbling of the urine. In enuresis in the narrower sense the urine is always passed in a stream. The diagnosis of the nature of the cause can often be made by a careful study of the case. Whether the disease is a persistence of the infantile condition, or has been acquired later, is readily shown by the history. The study of the urine will reveal the presence of any local disorders of the bladder or kidneys. Nocturnal enuresis occurring at long intervals is sometimes merely a symptom of nocturnal epilepsy, as pointed out by Pfister[1] and is not to be classed with the ordinary enuresis of childhood. Some other evidence of that disease can probably be discovered.

Treatment.—There is no certain cure, and the number of methods of treatment advised is large. Some do good in one case and some in another; and this is because the etiological agent varies with the individual. First of all the cause is to be sought and the proper treatment determined for it; although the habit of urinary incontinence seems often to continue after the removal of what might be called the original source. As in perhaps the majority of cases more than one factor is operative, or the true nature of these is not discovered, treatment must be varied, and directed in lines to meet a number of different conditions.

The *general hygiene* and *method of life* are to be carefully supervised. This applies to both the physical and the psychic condition. The child should pass much time in exercise in the open, life in the country being preferred. Sometimes a temporary change of residence benefits promptly. All mental overwork and all undue excitement must be avoided. The question of *school* is an individual one. Certainly the child with diurnal enuresis should not be sent, as his trouble makes him the object of ridicule by his school-mates, and this reacts upon his psychic state; and cases of nocturnal enuresis must avoid school also, if it is found to be too much of a strain upon the general health. Punishments are rarely permissible, as these only exceptionally do good and usually depress and frighten the patient and may make his state worse. On the contrary, the child must be encouraged in every way, and assured that he can and will recover; and perhaps be rewarded when he has been successful. In this connection the value of suggestion, hypnotic or of other nature, may be referred to.

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[1] Monatsschrift für Psychiatrie u. Neurologie, 1905, XV, 113.

It is certainly great in some instances. I have even known the suggestion that he shall not wet the bed, whispered to the child in a state of natural sleep, to be of service; and Dunham[1] reports a series of cases benefited by having the child repeat several times daily such sentences as "I am not going to wet the bed," and the like.

The *diet* requires a certain amount of regulation. It should be of an unirritating nature, with the absence of any article which is liable to produce indigestion or constipation. This must be studied for the individual child. The chief restriction is in the cases of nocturnal enuresis in which there is no highly acid condition of the urine present. Here no liquid of any sort should be ingested after about 4 P.M., the supper being entirely dry.

Sleep should be had in plenty, in well-ventilated rooms and with early hours of retiring; but, on the other hand, too profound a sleep is to be avoided. To accomplish this latter the child should not be allowed to become tired out before being put to bed; the bed-covering should be rather too light than too heavy; the bed should be hard rather than too comfortably soft.

The condition of the *general health* must be improved if necessary. Cool morning baths have a healthily stimulating effect. Massage is sometimes helpful. All such causative conditions as malnutrition and anemia, and any disordered state of the nervous system, must receive appropriate treatment. A cold douche to the spine before retiring is sometimes serviceable.

Local treatment is that applied to the removal of local causes of any sort, whether in the bladder itself or acting reflexly from some other region. Adhesions of the prepuce or clitoris may need to be broken up; phimosis may require circumcision; vulvovaginitis should be treated; and the local irritation from masturbation prevented by constant surveillance. Diseases of the rectum need treatment, including the removal of seat-worms. An evacuation of the bowels before the child goes to bed should be procured if possible, in order that fecal material in the rectum may not be a reflex stimulant for enuresis during the night. Urinalysis may show that pyelitis or cystitis exists and demands treatment, or other symptoms may suggest an examination for vesical calculus. When the urine is found highly acid, alkaline diuretics are needed or restriction of the amount of meat ingested, and water should be given in large amount rather than curtailed. In most cases, however, the urine is secreted in large quantity and is not too acid, and the amount of liquid taken needs to be diminished, as stated under diet, in order to lessen the fluid in the bladder. The child should be taken from bed several times in the night, if possible before the unconscious urination occurs. In this way the habit of continence is established. Further, in order to remove the pressure of the urine as far as may be from the sensitive trigonum and the neck of the bladder, there should be no pillows beneath the head and shoulders, the foot of the bed should be elevated, and the patient prevented from sleeping upon the back, this last being accomplished by tying a large empty spool over the spinal column. In the day-time the child should be taught to retain the urine as long as possible in order to accustom the bladder to its presence. This is of value both for the diurnal and the nocturnal forms of the disease. More distant reflex causes must be sought for and removed. Thus the operation for adenoid vegetations has been followed by the cure of enuresis in some instances.

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[1] Amer. Jour. Dis. Child., 1916, XII, 618.

Various sorts of local treatment of the bladder have been recommended in the effort to increase the tone of the sphincter muscles or to diminish the hypersensitiveness of the nervous apparatus. The passage of sounds of large size is one of these, and has proven effective in obstinate cases. The employment of the faradic current has also been strongly recommended, one pole being placed over the bladder and the other in the rectum or vagina, or sometimes in the urethra itself. The method should be reserved for intractable cases. Cauterization of the neck of the bladder, or, in girls, of the meatus has also been advised, as has massage of the neck of the bladder through the rectum or by deep pressure over the lower abdomen. Circumcision has been recommended even when no phimosis exists. Finally is to be mentioned the epidural injection of normal salt solution, as used by Cathelin,[1] with which good results have repeatedly been obtained. For girls past puberty and with enuresis persisting, the bladder may have become unusually small and unable to retain a normal amount of urine. In such cases daily dilatation by the injection of water may be tried. None of these more radical methods mentioned are to be employed except in obstinate cases which have resisted other forms of treatment.

Treatment with drugs is often very effective; often, like all other kinds, of no value whatever. That remedy should be chosen which counteracts the action of the cause. Thus strychnine is useful when there is a lack of tone in the sphincter; atropine when there is excessive irritability of the detrusor apparatus; iron or arsenic if there is anemia; alkalies when the urine is highly acid. As, however, it is often impossible to determine the exact cause or causes at work, the drug-treatment of the disease is to a large extent empirical and a great number of remedies have been advocated. Of all of them atropine is one of the most successful. A useful method is to prepare an aqueous solution of which each minim (0.062) contains 1/2000 grain (0.00003) of atropine. The initial dose at 5 or 6 years of age may be 4 minims, or even less owing to the idiosyncrasy to atropine sometimes encountered. This dose should be rapidly increased day by day, remembering that a large drop, from the edge of the bottle, equals approximately 1 minim. Children generally tolerate atropine in relatively large dosage, and enough must be given either to control the enuresis or to produce the physiological effect, especially the flushing of the forehead. Pushing the remedy beyond this amount is not advisable. It may be administered 3 times a day, or better, in cases of nocturnal enuresis, the first dose in the late afternoon and a second just before getting into bed. In my experience it is most important to hold the dosage at a maximum for a number of weeks, even if the result is good, and after that to diminish it very gradually; for if relapse occurs soon, as there is great danger of it doing if the drug is stopped, the atropine on the second trial will often fail to control the disease. The tincture of belladonna is more variable in its alkaloidal content and consequently less reliable. A standardized tincture may be given in corresponding dose (see table of doses, Vol. I, p. 229), 1 m. (0.062) of this equalling about 1/3000 grain (0.00002) of atropine. Bromide of soda may be administered in combination with the atropine with the same end in view, viz. the allaying of the excitability of the detrusor apparatus, and antipyrine is sometimes useful for this purpose.

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[1] Thèse de Paris, 1902.

In other cases where the trouble appears to depend upon the sphincter only, or in addition to the detrusor, strychnine may be given alone or combined with the treatment with atropine. It is indicated where the patient shows a general lack of tone, and is especially useful for diurnal enuresis. A child of 5 or 6 years may take from $\frac{1}{100}$ to $\frac{1}{60}$ of a grain (0.0006 to 0.0011) of strychnine 3 times a day. Tincture of nux vomica may be administered instead in proportionate doses (see table of doses, Vol. I, p. 230); 1 m. (0.062) equalling about $\frac{1}{400}$ (0.00016) grain of the combined alkaloids. Ordinarily about 1 minim may be given for every year of age. Tincture of rhus aromatica in doses of 5 to 30 minims (0.31 to 1.85) for children of 5 years has been sometimes useful in my experience. It is suitable in the cases of atony; and ergot acts favorably in the same class.

Among the various other remedies recommended and of service in some instances are humulus, valerian, zinc, chloral, hyoscine, phenacetin and thyroid extract. Some brilliant results with the last-mentioned have been reported by Williams.[1]

In *conclusion* the statements already made may be reiterated: that the causes are very varied and more than one is often present; that the treatment must be carefully selected according to the nature of the cause when this is discoverable; and that the disease is in a sense, a habit. Consequently the removal of the cause by no means immediately cures the disease, and efforts of all sorts must be persevered with until the habit is broken up.

TUBERCULOSIS OF THE BLADDER

This is a very uncommon affection in infancy and early childhood, but after this period may sometimes be found in combination with or be secondary to tuberculosis of other parts of the genito-urinary apparatus, especially the kidney; or to tuberculosis in some more distant region. It rarely occurs as a primary affection. It appears in the form of scattered, small greyish, or larger caseous nodules and of ulcers in the mucous membrane. Clinically the manifestations are those of chronic cystitis, with hematuria; although for a long period there may be no symptoms whatever. A positive diagnosis can be made only by the discovery of the tubercle bacilli in the urine or by cystoscopic examination.

MORBID GROWTHS OF THE BLADDER

These are of very uncommon occurrence in early life and may be primary or, oftener, secondary. Steinmetz[2] could collect but 32 primary cases. The most frequent growths are sarcoma and myxoma, and papilloma has also been observed. As the growth develops the symptoms of cystitis or of vesical calculus are liable to appear. Hematuria is more common than in ordinary cystitis. Palpation per rectum may aid in the diagnosis if the tumor is of considerable size; or a cystoscopic examination may be made. The growth may spread to the prostate or the vagina, or may give rise to pyelonephritis, hydronephrosis or peritonitis. Removal offers the only hope of cure in suitable cases; but is usually unavailing.

[1] Lancet, 1909, I, 1245.

[2] Deut. Zeitsch. f. klin. Chirurg., 1894, XXXIX, 313.

CHAPTER IV

DISEASES OF THE GENITAL ORGANS

ADHERENT PREPUCE

In the majority of new-born infants there is more or less adherence between the inner lining of the prepuce and the glans, and this may be looked upon as a normal anatomical condition. It is only as growth of the penis takes place that the adhesions disappear in the early months of life, and not infrequently they persist for a much longer time, even for some years, and are then to be considered pathological. The adhesions may be complete, or involve only the posterior portion of the glans. The smegma collects in quantities behind the corona and may be the cause of irritation producing priapism, painful urination, or balanitis from decomposition of the secretion.

The condition should be treated promptly in the new born by forcible retraction of the foreskin, followed by cleansing and the application of a bland ointment, and finally the drawing of the foreskin forward again. The mother should practice the retraction and washing daily, to prevent the re-formation of the adhesions; but no ointment should be applied after the torn surface has healed; unless this is necessary to permit of replacement of the foreskin in the normal position. The mother should be instructed never to leave the glans uncovered, lest paraphimosis develop, and she should see that the prepuce is always retracted sufficiently to expose the sulcus behind the corona. If the infant is allowed to live several months without the initial retraction, the adhesions may become so firm that separation with a blunt probe is required, using antiseptic precautions to guard against the development of inflammation.

PHIMOSIS

This consists in a narrowing of the prepucial opening, perhaps to an extent which may render retraction impossible. The condition is nearly always congenital, acquired cases being usually of temporary duration and dependent upon inflammation or edema of the prepuce. The opening may be so small that it is scarcely discoverable; or in rare cases there is none whatever. Adhesion may be combined with phimosis. The prepuce may be of normal length, or, much elongated and hypertrophied; but it is to be borne in mind that the condition of elongation (*redundant prepuce*) may be present unattended by phimosis.

Symptoms. When the opening of the prepuce is very small urination can be accomplished only by straining; the urine flows in drops or a very small stream; and, if adhesions are not present, the foreskin during micturition is ballooned by the urine beneath it. Hernia or prolapse of the rectum may be produced by the straining, or inflammation of the urethra, pyelitis, hydronephrosis, or pyonephrosis develop. All such serious complications are uncommon. In the majority of cases urination is not difficult, but the inability to keep the mucous membrane clean very commonly leads to posthitis or balanitis, with consequent dysuria through the swelling which results. The persistence of a constant degree of irritation readily leads to masturbation. Many other conditions are attributed to the presence of phimosis, among them enuresis, insomnia, night terrors, digestive disturbances, convulsions, epilepsy, and various neuroses. Doubtless some of these may be thus caused in occasional instances, but the influence of phimosis in this respect is certainly overrated. It is beyond doubt that even a very decided degree of phimosis may be present and produce no symptoms at all, or at the most those of slight local irritation.

The **diagnosis** is to be made only between preputial adhesions and true phimosis; and examination of the penis will readily settle this point.

Treatment.—Every case of phimosis should receive treatment of a nature to overcome the difficulty. In many cases forcible retraction or a stretching with dressing forceps is sufficient, or the preputial orifice may be widened by incision. When, however, a cutting operation is required circumcision is that usually to be preferred.

A few words may be said in this connection regarding the desirability of circumcision when no phimosis is present. A redundant prepuce, if sufficiently wide, does not call for it; since the foreskin may become none too long as the penis grows. Then, too, although circumcision removes the necessity of constant care for cleanliness, it is much disputed whether it is a procedure without subsequent disadvantages; and the operation is by no means a prevention of the development of the habit of masturbation.

PARAPHIMOSIS

In this condition the prepuce, which has been retracted beyond the corona, cannot readily be replaced. The circulation in the glans is interfered with by the constriction, and edema and bluish discoloration and even gangrene develop. The accident follows usually the retraction by the patient himself; or similarly, in the case of infants, that by the mother or nurse in attending to the cleansing of the organ. The symptoms, apart from the appearance already described, consist in pain and dysuria. By way of treatment cold compresses should be applied to reduce the swelling, and an effort made to draw the foreskin forward into the normal position. To aid in this the glans should be well oiled, steadily compressed, and a smooth instrument such as a grooved director or, in emergency, the rounded end of a sterilized metal hair-pin, slipped under the constricting skin. This serves as a starting-point for the replacement. If this procedure does not succeed, incision of the constricting ring of skin may be required. If the foreskin is unduly narrow, circumcision should follow.

STRANGULATION OF THE PENIS

A condition similar to that of paraphimosis or involving a larger portion of the penis is not infrequently produced by the patient tying a string around the organ or slipping a ring or other body over it. The treatment consists in division of the constricting body.

RUDIMENTARY PENIS

(Micro-penis)

The size of the penis varies greatly with the individual case. Sometimes in infancy the organ appears to be unusually small, due to a large deposit of fat in the mons and scrotum, which partially buries the penis from view. In other cases there is a decided retardation in the development of the organ, and I have known this to be the cause of great anxiety to the parents. As puberty approaches the penis usually enlarges rapidly and assumes its normal size. There are many cases of infantilism, however (see Infantilism, p. 530) in which development of the penis and of the body in general remains more or less permanently imperfect.

BALANO-POSTHITIS

Inflammation of the prepuce (posthitis) and glans (balanitis) is oftenest produced by phimosis, resulting in lack of cleanliness with an attendant retention and decomposition of smegma. In older boys it may follow injury caused by masturbation, or occur as a complication of urethritis, and a diphtheritic balano-posthitis is sometimes seen. The prepuce becomes red, edematous and itching, with the orifice narrowed, and the mucous membrane producing more or less purulent secretion. There is dysuria, and cystitis or hydronephrosis may result in severe cases. In ordinary cases the inflammation lasts but a few days.

Treatment consists in the injection of a strong solution of boric acid or one of potassium permanganate, 1:8000, under the foreskin in order to cleanse it thoroughly; after which a little boric acid ointment or zinc ointment may be worked carefully in with a probe. In severer cases cold compresses should be applied to reduce the swelling; and in the worst instances splitting or circumcising the foreskin is necessary.

MALFORMATIONS OF THE URETHRA

Hypospadias.—By this title is indicated the congenital malformation in which the urethra opens somewhere upon the lower surface of the penis instead of continuing to the end of this. The opening may be just behind the glans or in the body of the organ; while in the worst cases it is in the perineum, sometimes as a fissure, the scrotum being split into halves. As the penis is usually rudimentary in such cases and the testicles undescended, the appearance of the external genitals strongly suggests that of the female, and mistakes as to sex have often been made (*pseudohermaphroditism*). Treatment is surgical, and in many cases a plastic operation is very successful. Very rarely an analogous condition is seen in girls, the opening of the uretha being within the vagina, just behind the hymen, or still farther inside.

Epispadias.—In this very uncommon congenital anomaly the opening of the urethra is upon the dorsal surface of the penis somewhere in its length. It may be small, just behind the glans in the less severe cases, or may be in the form of a fissure the entire length of the penis, and is then combined with exstrophy of the bladder. The cases of slight degree can be cured by operation; the others present great difficulties.

Other Anomalies of the Urethra (*Atresia and Narrowing of the Urethra; Stricture; Diverticulum; Urethral Fistula; Vaginal Urethra*).—A congenital **atresia of the urethra** may range in degree from a mere closing of the meatus by adhesions, to complete obliteration of the urethra throughout its extent. Patulous urachus or exstrophy of the bladder will accompany the latter condition. In other cases there is a congenitally **narrow urethra,** perhaps only at the meatus; or there may be a cylindrical narrowing of some portion of the canal. Acquired stenosis of the urethra (**stricture**), may be the result, as in adults, of a gonorrheal urethritis, or rarely of cicatricial contraction depending upon other causes. A **urethral diverticulum** is a rare condition which may be congenital; or which may result from the constant difficulty in urination produced by stenosis, the presence of a calculus or a foreign body in the urethra, or trauma of some sort. It is characterized by distention of a part of the urethra during urination, which sometimes produces a discoverable swelling. The urine can be discharged from the sack by pressure. Bókai[1] was able to collect but 14 cases of congenital origin. **Urethral fistula,** of rare occurrence, may be of congenital origin the result of obstruction to the discharge of urine; or acquired through an injury done by a calculus or foreign body in the urethra, or by trauma of some other nature. The external opening of the fistula is in some portion of the penis or in the perineum, or rarely in other regions.

The treatment for these various conditions is surgical, and of a nature dependent upon the case.

URETHRITIS

This is an unusual affection in infants and not common in children of any age. Simple urethritis may be due to the extension of inflammation from an ordinary balanoposthitis; to irritation by highly acid urine; or to the introduction of foreign bodies into the urethra or other onanistic acts. Specific urethritis depends upon the action of the gonococcus. This latter, like the simple form, is seen much oftener in females and is then secondary to a gonorrheal vulvovaginistis; but it is surprising, with the frequency of this localization of gonorrheal infection, that urethritis does not occur more often. Mitchell and Quinn[2] collected 132 instances of gonorrheal urethritis in male infants reported since 1900. One of the youngest was a case recorded by them in a child of 3 months in my service in the Children's Hospital of Philadelphia. This case, it may be said, was the starting point for an outbreak of gonorrheal vulvovaginitis in the ward.

The **symptoms** consist in pain on urination, swelling and redness at the urethral orifice, and the discharge from the urethra of a small amount of muco-pus, which may be pressed from the canal or found in the urine. The symptoms are much more severe in the gonorrheal form. The **course** of the disease varies with the case. In the simple form the duration is usually short; in the gonorrheal cases much longer and liable to develop balanitis, arthritis and conjunctivitis. Stricture may be a sequel in boys. The **diagnosis** between the two forms of urethritis can be made with positiveness only by the examination of the secretion for the gonococcus.

Treatment in the simple form consists in the administration of alkaline diuretics and alkaline mineral waters, and in keeping the glans and prepuce disinfected, and the pus removed when balanoposthitis is the cause. In gonorrheal cases the same treatment is required as in the disease in adults. As there is great danger in this form of the infection being carried to other parts of the body, especially the eyes, a protecting antiseptic dressing should be constantly worn.

FOREIGN BODIES IN THE URETHRA; URETHRAL CALCULUS

Calculi which have descended into the bladder from the kidney may remain there and increase in size, or may pass out through the urethra in a short time. In such cases there are produced the symptoms described under Vesical Calculus (p. 203). Not infrequently the stone, if of considerable size or irregular in shape, becomes impacted in the urethra, oftenest in the membranous portion of the penis, although any part of the canal may experience it. Englisch[1] analyzes the reports of 405 such cases. Similarly, foreign bodies of various sorts may be introduced into the urethra, oftenest by boys during later childhood, and if of irregular or rough surface may remain impacted there.

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[1] Jahrb. f. Kinderh., 1900, LII, 181.

[2] Arch. of Ped., 1915, XXXII, 846.

The **symptoms** depend somewhat upon the size and character of the stone or of the foreign body introduced. At first there is more or less obstruction to the flow of urine, with straining efforts at urination. If the body is of a character to injure the urethra the evidences of a urethritis develop (p. 215), perhaps with periurethritis and urinary infiltration. All of these conditions increase the obstruction, until the flow of urine may be entirely cut off. In the cases of smooth calculi and foreign bodies the detrusor force of the bladder may succeed after several days in slowly propelling the object toward the meatus and finally expelling it; but very frequently surgical interference is necessary.

INFLAMMATION OF THE SCROTUM

This not unusual condition may be due to various causes. Most common is the irritation from neglect in changing the diapers and in cleansing the parts with sufficient frequency. From this cause the scrotum becomes intensely red, and, owing to the laxness of the tissues, much swollen. In other cases eczema is the cause, the rash then usually appearing upon the neighboring skin as well, and there is present the infiltration and often the vesiculation characteristic of the disease. Erysipelas may develop in the scrotum or spread to it from other parts. The swelling and redness are in this case more intense and fever develops. Rarely, as a result of erysipelas, urinary infiltration, perineal abscess, suppuration of the inguinal glands, or inflammation of the prepuce or of other neighboring regions, the inflammation of the scrotum becomes of a phlegmonous nature with great redness and hardness of the tissues, high fever, and severe constitutional symptoms. Abscess may form, or the skin may become of a bluish-red color and gangrene of more or less of the scrotum take place.

The **prognosis** of scrotal inflammation depends upon its nature. The phlegmonous process is always severe, since septic symptoms are liable to develop. **Treatment** in the erythematous cases consists chiefly of cleanliness and the keeping the scrotum dry with a talcum or zinc powder; or in cases where the inflammation is more severe, the application of wet antiseptic dressings. Eczematous and erysipelatous processes require the treatment appropriate for these diseases. Phlegmonous inflammation demands such surgical procedures as the local conditions indicate, combined with strongly supporting constitutional treatment and remedies to relieve the pain.

UNDESCENDED TESTICLE

(Cryptorchidism; Ectopia Testis)

Ordinarily the testis should descend into the scrotum in the 8th month of fetal life. Not infrequently (1 in about 1000 children, Willard)[2] arrest in the advance of one or both testes takes place, and the organ may remain permanently or for a time within the abdomen (cryptorchidism), or, more frequently, in the inguinal canal (*retentio inguinalis*). It is a common condition at birth, Ziebert[1] finding cryptorchidism on one side or both in 30 out of 102 new-born boys. If the testis has wandered into some abnormal position, oftenest in the perineum, the term *ectopia testis* is employed.

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[1] Arch. f. klin. Chirurg., 1904, LXXII, 487

[2] Surgery of Childhood, 1910, 213.

Symptoms.—Generally the testis retained in the inguinal canal will descend into the scrotum in the course of some weeks, or at least in the 1st year; but occasionally this may take place only after several years or even after adult life is reached. In cases of cryptorchidism no trace whatever of the testis can be discovered. In retentio inguinalis the organ is felt as a small, movable, oval body in the groin or above the scrotum, and may readily be mistaken for hernia. The difficulty of diagnosis is increased by the fact that the testis in this situation is accompained by hernia in half or more of the cases. The exposure here renders it readily subject to trauma, with resulting pain and tenderness from inflammation. Sometimes symptoms are extremely severe and the strangulation of a hernia is suspected. A testicle remaining persistently out of place finally becomes atrophic and functionally useless, and does not secrete semen. In cases of long-continued double cryptorchidism, the patient will probably be sterile, and occasionally the development of masculine characteristics is interfered with.

Among the **complications** are hernia, the development in the testis of malignant growths, hydrocele, and torsion of the spermatic cord with gangrene.

Treatment.—This is not required in infancy, as the probability is that the testis will descend of itself. In early childhood daily manipulation may be tried in the effort to bring the organ into the scrotum. By the age of 8 or 10 years operation should be performed in bilateral cases with the intent of transplanting the testis into its proper position. In unilateral cases operation is not so imperative, but is certainly called for if hernia is present or if there is persistent pain. Removal of the testis should not be performed unless its structure has been destroyed by degenerative processes and the effect of its internal secretion removed. Replacement into the abdominal cavity is to be made in cases where the testis cannot be transplanted and when frequent pain is produced by the exposed inguinal situation.

ORCHITIS. EPIDIDYMITIS

An **acute orchitis** is occasionally seen in children as a result of trauma, rheumatism, and infectious fevers, especially mumps. (See Epidemic Parotitis, Vol. I, p. 499.) Gonorrhea is more likely to produce an **epididymitis** than affect the testis itself. Inflammation due to this cause is of rare occurrence in early life, Mitchell and Quinn[2] being able to collect but 3 cases reported since the year 1900, to which they added a 4th instance in an infant of 3 months under my observation. There is redness of the scrotum with pain, tenderness, and swelling of the testicle, and fever. The prognosis is usually favorable unless suppuration occurs, and the course is short. There is a possibility, however, that orchitis following mumps may result in atrophy of the testicle. In the way of treatment the patient should be kept in bed, with the testicle elevated, and cold, wet dressings applied.

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[1] Beiträge z. klin. Chirurg., B. II, H. 2. Ref., Langstein, in Pfaundler and Schlossmann, Handb. der Kinderh., 1910, IV. 116.

[2] Arch. of Ped., 1915, XXXII, 846.

Syphilitic orchitis or **epididymitis** runs a more chronic course. Occurring to a slight degree it is frequent in cases of hereditary syphilis. The testicle is enlarged, painless and very hard, but does not suppurate. The lesion consists usually in an interstitial inflammation affecting either testis or epididymis, but occasionally gummata are found. More or less atrophy of the gland follows. The diagnosis of this form rests upon the unusual hardness and the presence of manifestations of syphilis elsewhere in the body.

MORBID GROWTHS OF THE TESTIS AND PROSTATE GLAND

Neoplasms of the **testis** are infrequent in early life. The decided majority are malignant in nature, carcinoma being more common than sarcoma. Steffen[1] could collect but 13 instances of malignant new growths of the testis. Enchondroma and teratoma are very rare. Morbid growths may be seen in earliest infancy or even be congenital. Usually only one testis is affected. There are no symptoms at all at first, but later the general health suffers, and there may be dull pain and a dragging sensation in the gland. Usually the scrotum becomes involved and the growth, at first smooth and hard, becomes nodulated, the mass softens, and ulceration takes place. Metastasis may occur. Early operation may accomplish a cure, but as a rule the prognosis is unfavorable and recurrence is very liable to take place.

Malignant tumors of the **prostate gland,** usually carcinomatous, are still less common, and Steffen collected but 6 cases. Interference with urination and pain are among the early symptoms. Secondary enlargement of the inguinal glands is frequent. As the tumor grows the pelvic organs may be displaced and the general health deteriorates. Metastasis frequently occurs. The course is rapid and death takes place in a few months. Operation is valueless.

HYDROCELE

This quite common disorder consists in an accumulation of serum around the testis or the spermatic cord. It may be congenital or acquired; the latter less frequent in children than the former, being produced by pressure, inflammation or injury. Several varieties are encountered:

1. Communicating Congenital Hydrocele.—In this form of hydrocele, common in the 1st year of life, the extension of the serous membrane from the peritoneal cavity has never become closed above and a communication exists, either freely or through a very small opening. This permits the accumulation of fluid to be present in the tunica vaginalis at birth, although it may not become evident until later The fluid can be readily reduced.

2. Communicating Hydrocele of the Cord (*Communicating Funicular Hydrocele*).—In this comparatively uncommon variety, also of congenital origin, the pouch containing the fluid is shut off from the testis below, but communicates with the peritoneal cavity above. Reduction can be obtained as in the former variety. The hydrocele is situated in the line of the spermatic cord above the scrotum.

3. Infantile Hydrocele (*Hydrocele of the Tunica Vaginalis Testis*).—This title is applied to a collection of fluid in the tunica vaginalis, the communication with the peritoneal cavity having been cut off, making it irreducible. It is one of the commonest varieties of hydrocele. The fluid is situated within the scrotum.

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[1] Die maligne Geschülste im Kindersalter, 1905, 62.

4. Encysted Hydrocele of the Cord.—Here the funicular process of the peritoneum is closed both above and below, forming a sac containing liquid in the course of the cord, with the testis below it.

5. Hydrocele of the Canal of Nuck.—This is a variety occasionally seen in females and develops in the peritoneal process which in the fetus surrounds the round ligament.

Symptoms and Diagnosis.—**Communicating congenital hydrocele** forms a swelling extending throughout the whole length of the cord to the lower part of the scrotum. The testis is at the posterior part of this and rather high. As the fluid is readily reducible if the patient is on his back, either spontaneously, by gravity, or by pressure if the communication is small, it may easily be mistaken for hernia, with which it is, indeed, often combined. In contrast with hernia the swelling is, however, dull on percussion, more fluctuating, translucent, and does not under manipulation slip back suddenly into the peritoneal cavity with a gurgling sound.

Communicating hydrocele of the cord forms a small, elastic, fluctuating, elongated swelling above the scrotum. It is reducible like hernia, but is distinguished in the same way as in the form just described. After reduction the cavity soon fills again. The testicle is in the normal position near the bottom of the scrotum. Hernia may complicate the disease.

Infantile hydrocele presents an accumulation of fluid filling the scrotum and forming an oval, elastic, fluctuating, tense, translucent sac. The spermatic cord and ring can be distinctly felt above it. The testis is posterior and rather high in position. The swelling is irreducible and not affected by cough or position, and is in other respects distinguished from hernia by the characteristics described for the first variety of hydrocele.

Encysted hydrocele of the cord forms a small cyst, spindle-shaped or globular, suggesting often an undescended testis, small hernia, lymphatic gland, or a tumor of some sort. From the testis it is distinguished by its translucency and by the finding of this organ in its normal position in the scrotum; from a lymphatic gland or tumor by its elasticity and translucency; and from hernia by the same qualities together with its irreducibility and dullness on percussion.

It is to be remembered, however, that in hernia the intestine may sometimes be distended by gas and have a somewhat translucent character. Repeated examinations will, remove the doubt in diagnosis. Two forms of hydrocele may be combined, or the sac of a congenital hydrocele with a large opening into the peritoneal cavity may contain fluid at one time and intestine at another.

Prognosis and Treatment.—The prognosis in early life is good. The majority of cases will recover of themselves, and this is especially true of the communicating forms. If not, the wearing of a truss will usually effect a cure by producing a mild obliterating inflammation. Encysted hydrocele of the cord may be painted with tincture of iodine. For the infantile form the application has been advised of adrenaline, lead-water, a saturated solution of magnesium sulphate, or other lotion. Yet one must not forget that the tissues of the scrotum are very easily irritated and that even dangerous inflammation may be set up. It is not often that operation of any sort is necessary in early life, as the majority of hydroceles will disappear of themselves in the course of a few weeks or months. The simplest operation is aspiration with a small needle and syringe, and this is generally all that will be required.

TUBERCULOSIS OF THE MALE GENITAL ORGANS

That **tuberculosis of the prepuce** may be acquired through ritual circumcision has been especially emphasized by Holt,[1] who collected 41 cases from medical literature, including 1 of his own. In all of them ulceration developed. It is known that at least 16 of these cases died later from a general tuberculosis.

Tuberculosis of the testis was seen by Jullien[2] 17 times in 5566 sick children under 13 years of age. Out of 51 collected cases up to the age of 17 years reported by Kantorowicz,[3] 21 occurred in the first 2 years. Although, therefore, encountered oftener in the first 2 years than at other periods of early life, it is less frequently seen in children than in adults. Congenital cases have been reported, as by Dreschfeld[4] and others. Trauma is sometimes a predisposing cause. Oftenest only one testicle is involved. The lesion may be primary or secondary to tuberculosis elsewhere. It appeared to be primary in 46 of Kantorowicz's 51 cases.

The onset may be slow or rapid. The testis with the epididymis becomes enlarged and hard, and later in part adherent to the scrotum, which assumes a bluish-red color at this position. Still later the process may either undergo absorption, or may soften, break down, and discharge pus with cheesy masses. This unfavorable termination is especially liable to occur in infancy, but the tendency to the persistence of a fistula is less often seen than in adult life.

The course of the disease is often much protracted and development of tuberculosis in other parts of the body sometimes follows. The prognosis on the whole is better than in adult life, both as regards local recovery and extension to other regions.

Treatment is constitutional as well as local; a suppurating testis being removed only if other means fail.

DISORDERS OF THE BREASTS

The frequent presence of milk in the breasts soon after birth, and the very common development of **mastitis** at this age, either in male or female children, has already been described under Diseases of the New Born (Vol. I, p. 295). Later mastitis may occasionally result from trauma or some infection. **Absence of the breasts** is extremely uncommon and is usually combined with other malformations. **Supernumerary breasts** are more frequently seen. They may occur in either sex, oftenest in the trunk and just below the normal breast; or sometimes in the axilla or in other parts of the body. Four breasts is the most frequent number. The condition may be hereditary. **Early enlargement of the breasts,** apart from the temporary condition in the new born alluded to, is seen oftenest as an accompaniment of precocious menstruation. It occasionally, however, occurs independently of this, either in the female or male.

Tumors of the breast in early life are entirely exceptional. Those reported comprise, among others, angioma, fibroadenoma, carcinoma, sarcoma, and cystic growths. The subject has been studied especially by Jopson and Speese,[5] who could collect but 30 instances of mammary tumors in children, 21 of these being of a benign nature.

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[1] Jour. Amer. Med. Assoc., 1913, LXI, 92.

[2] Arch. gén. de méd., 1890, CLXV, 1, 420.

[3] Dissert. Berlin, 1893.

[4] Brit. Med. Journ., 1884, I, 860.

[5] Annals of Surgery, 1908, Nov.

MALFORMATIONS OF THE VULVA AND VAGINA

An **atresia** of the vulva is a rare congenital occurrence. It consists in an adhesion of the surfaces of the greater or lesser labia. It may be only an epithelial adhesion or may be a firmer union, which, if not complete, may cause a retention of mucous secretion, and later of the menstrual discharge, and, if complete, will also interfere with the passage of urine. A similar result in the retaining of menstrual discharge is produced by an **imperforate hymen.** Sometimes there is an **absence of the vagina** through a considerable part of its extent. In any of these conditions operative procedures are required. Adhesions between the labia often only need to be broken up by the employment of a probe, as in the analogous condition of the prepuce.

Malformations of the vulva or vagina are often associated with those of the rectum and anus. Sometimes there is an abnormal opening existing between the rectum and vagina, and, if this is combined with atresia of the anus, the feces are discharged by way of the vagina. In other cases the reverse is true, and with atresia of the vulva any discharge from the vagina, uterus, or bladder passes off by way of the rectum.

AFFECTIONS OF THE CLITORIS

The anterior portions of the labia minora unite to form the prepuce of the clitoris. There is frequently **adherence** between the labia and the clitoris, analogous to the condition seen in males. As a rule no symptoms are produced, but sometimes an undue irritation arises from the retained smegma, with a consequent reflex nervous condition or masturbation. With a probe the adhesions can be readily broken up. In pseudo-hermaphroditism, especially in connection with diseases of the adrenal bodies or pineal gland, there is very decided **hypertrophy** of the clitoris present, to such an extent that it may resemble a small penis without any meatus. **Congenital fissure** is a rare condition usually seen in combination with fissure of the urethra.

VULVOVAGINITIS

This disease is one of the most common and widespread of the affections of female children. It occurs chiefly in two forms (1) Simple Vulvovaginitis, and (2) Gonorrheal Vulvovaginitis. The latter, once thought to be uncommon, is now known to be by far the most frequent form.

1. Simple Vulvovaginitis (Non-gonorrheal). Etiology.—Any age may exhibit it, even the new born, as emphasized by Epstein.[1] At this early period it appears to be the result of the normal desquamative process taking place in the vagina, rendered worse by neglect and consequent development of a catarrhal inflammation. The disease is, however, more frequent after infancy has passed. It is then caused by uncleanliness; traumatism from various causes including, possibly, masturbation; eczema; thread-worms in the vaginal canal; anemia and other debilitating influences; and the various infectious fevers. The vesicles of varicella may sometimes appear upon the mucous membrane of the vagina and give rise to vaginitis. Germs of various sorts, especially cocci, including the pneumococcus, but other than the gonococcus, are the active agents, and there is good reason to believe that this disease is somewhat infectious and transmissible from child to child.

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[1] Arch. f. Dermatol. u. Syphilis, 1891, XXIII, Ergänzungsheft, II, 3.

Symptoms.—The vulva, vagina, hymen, orifice of the urethra, and the cervix of the uterus are red and swollen, but usually only to a moderate degree. There is a vaginal discharge which is scanty, or in severe cases abundant; and is either serous, thin, and milky; or thick, yellow, distinctly purulent, and often offensive in odor. In severe cases the cutaneous covering of the vulva and thighs may be excoriated, and urination and walking may be painful. Microscopic examination shows pus-cells and bacteria in varying numbers.

Course and Prognosis.—If the causative factor can be readily removed, the prognosis is good, and recovery will take place in a few weeks under treatment; but often in debilitated subjects the discharge continues for a long time in spite of this.

Diagnosis.—The simple form differs from the gonococcic chiefly in the greater involvement of the vulva; the absence or slight degree of contagiousness; and the absence of complications and of the gonococcus in the discharge. To this last there are numerous exceptions, and cases of gonorrheal infection, if of long standing, may fail to show the gonococcus, and it may require culturing to reveal them, as shown by Welt Kakels[1] and others. The employment of the Gram stain is necessary to differentiate, inasmuch as cocci of other kinds than the gonococcus may be found within the pus-cells.

Treatment.—This consists primarily in the search for and the removal of the cause. Cases dependent upon debility demand tonic remedies, and sometimes change of air is very efficacious. Locally it is important to keep the parts scrupulously clean by frequent washing with weak antiseptic lotions, such as a saturated solution of boric acid; or one of permanganate of potash (1:8000) or of corrosive sublimate (1:10,000). More resistant cases require vaginal injections of these, given with a small catheter passed within the hymen. A solution of argyrol (5 per cent. to 20 per cent.) or of alum (saturated) is often also useful. An ointment of boric acid or one of oxide of zinc may be smeared over the skin on the exterior of the vulva to protect this region. These are made more adherent if mixed with a certain amount of starch, as in Lassar's paste (zinc oxide ½ oz. (16); amylum ½ oz. (16); petrolatum 1 oz. (31)).

2. Gonorrheal Vulvovaginitis. **Etiology.**—Girls of any age, even infants or the new born, are very subject to this disease. Its frequency is very great. Holt[2] found that there were rarely less than 5 or 6 cases in an average of 125 applicants for admission to the Babies Hospital of New York. A similar incidence (5.3 per cent.) is reported by Taussig[3] for St. Louis. It is especially common in infancy and in the first 5 years of life. Of W. Hamilton's[4] 344 cases, 151 (44 per cent.) were under 5 years of age. All that is needed to produce it at any age is the entrance into the vagina of the gonococcus. The method by which this is brought about is, in most cases, not discoverable; and is in hospital wards and institutions so difficult to prevent in spite of the greatest care, that from a single case the disease often rapidly spreads to a large number of the other inmates and becomes epidemic. The great majority of cases of leucorrheal discharge seen in hospital practice are gonorrheal in nature. There is hardly any other infection occurring in institutions for children the control of which is so discouraging to the physician. In the case of infants, the disease may be acquired during the process of birth, or later from a mother with a gonorrheal discharge; or may be spread in various other ways, as by the hands or garments of the nurse or other persons; the use of infected bath-tubs, diapers, bed-clothing, wash cloths, thermometers, or towels; or be acquired from the floor upon which the child may crawl and sit. It is commoner among the poor on account of the lesser care taken in the way of cleanliness; but it is also not infrequent under what appear to be the best hygienic surroundings. In girls past the period of infancy other methods of infection are to be considered as well as some of those mentioned. Prominent here is the sleeping with a mother who has a leucorrheal discharge; in reality gonorrheal, but unrecognized. Contamination by soiled water-closet seats is doubtless a frequent method in school-life, and among other causes is the public bath. Skutsch[1] reported 230 cases developing in 14 days arising from the use of the public baths. Handling of the genitals by some other child who has the disease is another method occasionally encountered, and much less frequently rape or attempted coitus with a boy suffering from gonorrheal discharge. The contracting of the disorder by any sexual act is comparatively uncommon in children.

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[1] New York Med. Journ., 1904, LXXX, 740.
[2] New York Med. Jour., 1905, LXXXI, 521.
[3] Amer. Jour. Med. Sci., 1914, CXLVIII, 480.
[4] Jour. Amer. Med. Assoc., 1910, LIV, 1196.

With this is said by no means all in the matter of etiology and the relationship of the gonococcus to the disease. There is no question that the acting organism is one which in appearance and on culture has all the characteristics of the gonococcus as seen in the adult. Yet the course of the disease in infancy and early childhood as compared with that of later years: the lesser intensity of the inflammation; the infrequency of complications; and the failure of nurses to contract the disease from patients force the conclusion either that the virulence of the germ as it occurs in early life is greatly modified, or that it is in reality different in certain respects from the gonococcus of the adult, at least in the majority of instances. That the latter is very probably the case has been indicated by agglutination and complement-fixation tests which have shown (Pearce)[2] that two types of the organism exist; one, the adult type, capable, for instance, of producing ophthalmia as a complication, the other, much the most frequent type in early life, never doing this.

Symptoms.—These are largely similar to those of the simple vaginitis described. In the milder cases there may be a very small amount of thin discharge, and the disease may be overlooked if precautionary measures are not taken in hospital epidemics to examine cover-glass preparations from all the female patients. From this degree the discharge may vary up to an abundant, yellow, purulent secretion. The mucous membrane throughout the vulva and vagina are inflamed as in the simple catarrh, although in the severer cases to a greater degree. Ordinarily there is little or no discomfort and no fever; but in severe cases there may be rise of temperature, pain on urination, excoriation of the skin of the vulva and thighs by the discharge, and various complications.

Course and Prognosis.—Although seldom a serious affection in early life, the disease is a most unpleasant one, very tedious in recovery, and a source of great anxiety and often of unwarranted mortification to innocent parents. The disease appears to recover of itself as time passes, and apparently leaves no permanent after-effects. From 6 to 10 weeks is required for the cure of the acute stage, but months or even years may elapse before complete recovery takes place. It is even claimed by some writers that the disease is incurable but this view is hardly tenable. That permanent recovery does occur is proven not only by clinical experience, but by means of the complement-fixation test.

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[1] Inaug. Dissert., Jena, 1891. Abstr. in Arch. f. Derm. u. Syphilis, 1891, XXIII. 672.

[2] Journ. Exper. Med., 1915, XXI, 289.

On the whole, the disorder is to be looked upon as a most disagreeable misfortune rather than a dangerous or even serious malady. Everything seems to indicate that, when acquired early, it rarely if ever produces the lasting damage which gonorrhea contracted in adult life is so liable to leave behind it. Were it otherwise, with the enormous prevalence of the disease, we should, for instance, find sterility in women of the poorer class many times more frequent than we know to be the case. It is certain that relapses are very common, and that the disorder may pass into a latent state, which may last for months or even over a year, in which no discharge occurs and no gonococci can be found, although at last a recurrence takes place. It is due to these latent cases that epidemics break out in hospital wards, in spite of care in guarding against the admission of a patient with the disease; infection having been disseminated even before discovery of the original case is made.

Complications.—These are not of frequent occurrence, being very much less often seen than in adult life, and chiefly after the period of early childhood. Swelling of the inguinal glands may rarely take place, and, very exceptionally, a suppurating bubo may form. Sometimes the urethra and bladder are involved in the process, and a gonorrheal cystitis or pyelonephritis may develop. This is of infrequent occurrence. Cystitis was seen in 5 of 74 cases as reported by Spalding.[1] This is a much larger percentage than is ordinarily experienced, if one bases the diagnosis at all upon symptoms connected with the bladder. Proctitis is likewise an occasional complication which disappears with recovery from the vulvovaginitis. Flügel[2] found it in 20 per cent. of his cases; but such a high percentage is unusual. It may possibly be developed by the rectal employment of the thermometer in cases of gonorrheal vulvovaginitis. It is very exceptional in infancy that the disease extends upward beyond the cervix uteri, producing endometritis, salpingitis, or pelvic peritonitis, and even in childhood the occurrence is not common. I have seen but 2 instances with pain suggesting mild involvement of the pelvic peritoneum, and these were in subjects beyond the period of infancy. Others, however, have found it more frequent, and sometimes widespread in the general peritoneum; when it constitutes, of course, a serious affection.

One of the most dangerous complications is gonorrheal ophthalmia. Sheffield[3] found it in 7 (4.7 per cent.) of his 148 cases. It is surprising that it is not encountered more frequently. In my own experience the incidence is very much less than this. Another complication is gonorrheal arthritis, involving one or more joints. This occurred in 4 of Spalding's[4] 74 cases. The process is of a septic nature. (See Arthritis, p. 439.) General septicemia is a rare condition, as is involvement of the endocardium, pericardium or meninges. The infrequency of complications is shown by the analysis by Gittings and Mitchell[5] of 188 instances of these, collected from the reports by different observers. Among the conditions mentioned are: arthritis 3 cases; general peritonitis 2; ophthalmia 4; procitis 3; and urethritis 1.

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[1] Amer. Jour. Dis. Child., 1913, V, 248.
[2] Berl. klin. Wochenschr., 1905, XLII, 325.
[3] New York Med. Rec., 1907, LXXI, 767.
[4] *Loc. cit.*
[5] Amer. Jour. Dis. Child., 1917, XIII, 448.

Diagnosis.—The diagnosis of gonorrheal vulvovaginitis is probable if the secretion is very purulent; but as a matter of fact it is safest to consider all leucorrheal discharges, especially in infancy and early childhood, as gonorrheal until proven otherwise. The only positive diagnostic sign is the discovery of Neisser's gonococcus within the pus cells, and the decolorization of the germs when stained by Gram's method; and sometimes even culturing or the complement fixation test is necessary in order to insure against error. One negative result is not sufficient, and the examination should be repeated. After cleansing the external genitals the labia should be separated and the secretion obtained from well within the vagina, using a cotton swab or dull metal instrument, such as a groove director or a platinum loop, and employing a small speculum if necessary. When this procedure fails to reveal the germs, ½ fl.oz. (14.79) or more of a 1 : 5000 solution of mercuric chloride in normal salt solution should be injected into the vagina, while the child is on her back with the hips elevated. An all-rubber eye-and-ear syringe may well be employed for this purpose. A smooth glass rod should now be inserted and the mucous membrane gently rubbed with it. The fluid is then withdrawn with the syringe, centrifugated, and the sediment stained and examined (Norris).[1]

Treatment. *Prophylaxis.*—What has been said above regarding the lack of seriousness of the affection in the very large majority of cases in no way militates against the taking of every possible precaution against its dissemination; since the disorder is one which no one would lightly allow an innocent infant or little girl to acquire. Attention to the minutest details is required to stop the spread of the disease from an affected child to others. This is especially true of hospitals, schools, and other institutions for children. Before entering into a hospital for children, every female patient should have a smear, or still better a washing, of the vagina made and the secretion examined for the gonococcus. If the result is positive the child should be admitted to a special ward. Similarly every child already in the hospital who develops a leucorrheal discharge should be examined in the same way, and if gonococci are found, removed to a special ward, and attended by a nurse who shall not enter the general ward. This is, however, not a sufficient protection, owing to the latent period which the disease exhibits; and the prevention of outbreaks will be aided by having repeated routine examinations of all female patients performed at least once a week. This separation of patients and their attendant nurses needs to be more thorough than in almost any other infectious malady.

The employment of various other measures to prevent infection are important; and particularly so when for any reason complete quarantine cannot be established. The clothing, towels, and other washable articles of all infants and children in the hospital, even when not suffering from the disease, should be soaked in a disinfectant solution and then boiled before allowing them to go into the general laundry. Single-service diapers of cotton, absorbent gauze and paper should be employed instead of the ordinary textile ones, and destroyed after use. In all cases with the disorder a protective dressing should be worn over the vulva to prevent the soiling of the clothing, and the infection of the child's hands. There should be individual toilet articles for each female in the ward, and an individual thermometer. The employment of bath-tubs is dangerous even when no case has yet developed, and a spray bath is to be preferred in the case of infants. When bath-tubs are required for the purpose of hydrotherapy, thorough disinfection of these should be attempted, with steam if practicable. The nurse attending a case of this disease should wear rubber gloves when waiting upon the patient, and carefully disinfect these before using them and after their removal. She should also protect her clothing in order to prevent possible carrying of the germs in this way. The gonococci are not difficult to destroy if they can be properly reached, but are disseminated with such extraordinary ease that in spite of all precaution, the checking of an epidemic is difficult. I have seen this extension take place in spite of separate nurses, rubber gloves, separate rooms, single-service diapers, individual thermometers, and the like. Apparently the only efficient method is the removal of affected cases to parts of the building where those in charge of them cannot come into even remote association with other children; and even this is not a sure method, owing to the fact that the disease is doubtless spread from a latent case before the existence of the infection is discovered.

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[1] Jour. Amer. Med. Assoc., 1915, LXV, 327.

In practice outside of hospital wards the same preventive measures should be followed as far as possible when the disease has already appeared in one child of a family. The affected child should wear an antiseptic occluding pad; have individual bed, bed-linen, and toilet-articles; all washable articles should be disinfected promptly by carbolic acid and subsequent boiling; careful precaution should be taken against the contamination of toilet-seats and bath-tubs; and the child should come into as little close contact as possible with other children of the family. There seems no doubt that the disease is disseminated to a large extent by school-life, and probably chiefly through the lack of cleanliness in the water-closets and out-houses. These should always be furnished with a U-shaped seat, and this kept as clean as possible.

The question of an educational campaign is a perplexing one. That mothers, school teachers, hospital authorities, nurses, and all coming into contact with many children should be taught the nature of the disease, its tendency to spread, and the proper methods of prevention is certainly indisputable. On the other hand, when dealing with a laity so susceptible to misunderstanding and excessive timidity, that one mother fears even to telephone another whose child has an infectious fever, it is equally evident that great caution must be observed lest more harm be done than good in the community. The propaganda against vulvovaginitis emanating from the medical profession have not always been conducted in a way to avoid this. There is not as yet any satisfying proof that the disease is sufficiently serious in its immediate or ultimate effect to warrant repeated, systematic vaginal examination and studies of the secretion from all female school children: the forbidding the affected child attending upon public school and the reporting of the case; or in any other way publicly branding an innocent little girl as the subject of a noisome venereal disease. The measures proposed in many quarters are far too radical.

Treatment of the Attack.—In the line of cleanliness, the methods to be employed have already been described for non-specific vulvovaginitis. The vulva and vagina should be douched twice daily with a saturated solution of boric acid or one of potassium permanganate (1 : 8000), or corrosive sublimate (1:10,000), using a small catheter to ensure that the fluid passes high into the vagina. This should be followed by the instillation of a 25 per cent. solution of argyrol or 1 per cent. to 3 per cent. of protargol or other preparation of silver. To accomplish this satisfactorily the hips should be well elevated, and the thighs bent over on the abdomen in order to keep the remedy in contact with the mucous membrane for a considerable time. This should be followed by the application of a protective occluding pad. I have also used with satisfaction narrow vaginal suppositories of argyrol of 25 per cent. strength. These have the advantage that the contact is of longer duration.

Treatment should be persisted in until all discharge has ceased, and the gonococci have disappeared; and even after this it is safer to continue applications once a day or at least every other day for a considerably longer time, and to follow precautions against infection of others. Several negative smears should be obtained before the case is called cured. Even then this judgment must be expressed with considerable hesitation, owing to the tendency to relapse after a period of perhaps several months; and occasional smears should still be taken at intervals.

Vaccine Treatment.—The hypodermic injection of gonococcus vaccine in the treatment of this disease has enthusiastic followers, while there are others who have obtained no benefit whatever with it. W. Hamilton[1] reports 60 per cent. cured in 260 cases treated by irrigation, and 90 per cent. of recoveries in 84 cases receiving vaccines. The average duration of the disease in the first series was 10.1 months, and in the second series 1.7 months. Finger,[2] on the other hand, concludes that there is never more than an improvement even with large and repeated doses of vaccines. The final place of the procedure in therapeutics has, therefore, still to be determined. It certainly appears to be successful in cases of arthritis. It should be tried in the obstinate cases, on account of the surprisingly good results which have been reported in some instances. Either autogenous vaccines may be employed, or a polyvalent one from several stocks. The injection may be given once or twice a week. The dosage recommended varies greatly; from 5,000,000 to 50,000,000 for a single dose, the number being increased rapidly to doses of 400,000,000.

3. Herpetic Vulvitis (*Aphthous Vulvitis*).—Accompanying the development of herpes upon the cutaneous surface of the vulva, and often upon the perineum, groin, and inner surfaces of the thighs, there may be found upon the vulvar mucous membrane an eruption of numerous small vesicles becoming placques, and in 2 or 3 days superficial ulcers. The same series of changes may occur in the cutaneous lesions. The disease occurs most frequently in infancy or early childhood, in unhealthy or neglected subjects, and often after infectious diseases, especially measles. It is sometimes seen also in cases of nephritis. The ulcers may coalesce and cause pain and itching and a vaginal discharge. There is some fever, but there is little, if any, glandular involvement. Under treatment the disease disappears in a few days, although it is possible for gangrene of the vulva to develop as a sequel.

Treatment consists in keeping the parts very clean, and the application of a powder of iodoform, boric acid, or of a combination of oxide of zinc and starch. In other cases these remedies in the form of an ointment are to be preferred.

4. Phlegmonous Vulvitis. (*Abscess of the Vulva.*)—This is the result of trauma of various sorts, erysipelas, or the extension of inflammation from a vulvovaginitis. The external labium on one or both sides becomes red, swollen, indurated and tender, and restlessness and fever are present. Usually the inflammation advances to the production of pus. **Treatment** consists in the application of cold compresses, or in incision if abscess forms.

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[1] *Loc. cit.*

[2] Wien. med. Wochenschr., 1914, LXIV, 862.

5. Gangrenous Vulvitis (*Noma pudendi*).—By this title is designated a gangrenous condition analogous to the noma seen in the mouth. It occurs in cases of malnutrition especially after some of the infectious diseases, chiefly measles or erysipelas, or may follow aphthous inflammation of the labia. In a case reported by Rach[1] numerous spirochetes and fusiform bacilli were found. A swollen, tense, indurated, shining, dark-red area appears upon one of the external labia. This soon becomes dark-blue and then black in color, breaks down, and discharges an offensive gangrenous material. In the severe cases the disease spreads with great rapidity, involving more or less of the whole vulva, and extending to the vagina, perineum, mons veneris, or other neighboring parts. The constitutional symptoms are those of extreme prostration, with pain and fever, and death nearly always results. In the milder cases the gangrenous process is limited to a small area, the slough is cast off, and an ulcer remains which heals with more or less cicatricial deformity. Treatment must be instituted early to be of any avail, and consists in prompt, thorough incision followed by cauterization.

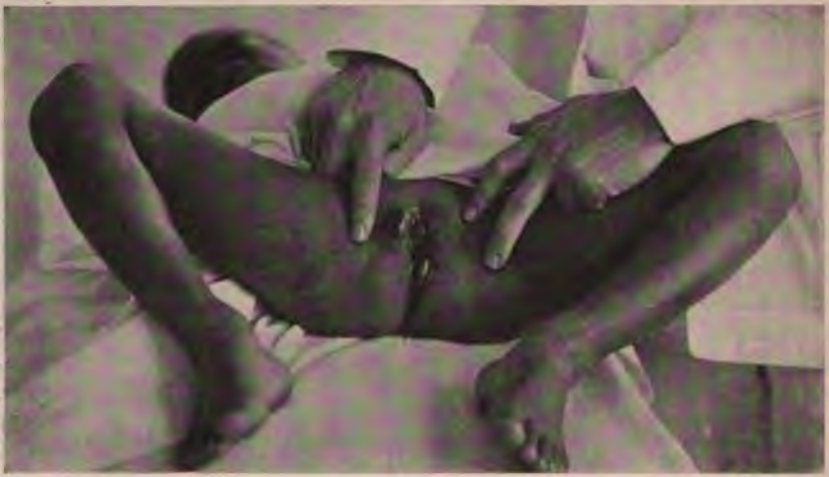

FIG. 323.—WARTY GROWTHS OF THE VULVA.
Courtesy of Dr. Harry Lowenburg.

6. Diphtheritic Vulvovaginitis.—Occasionally diphtheria may show itself upon the female external genitals. In may be solely here, or exist as a complication of diphtheria of the nose and pharynx. The labia are swollen, red, and covered on the mucous surface and sometimes on the cutaneous also by a deposit of greyish or yellowish pseudomembrane. Local cleansing and antiseptic treatment is required, in addition to the administration of antitoxin.

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[1] Jahrb. f. Kinderh., 1911, LXXIII, 231.

MORBID GROWTHS OF THE VULVA AND VAGINA

These are of very exceptional occurrence, and are generally encountered very early in life, or still more frequently occur congenitally. Sarcoma is the form most often seen. Steffen[1] collected from medical literature 15 cases of malignant vaginal tumors in children, in addition to 6 others affecting both vagina and uterus. Mergelsberg[2] has increased the list to 37 cases. A benign growth is also occasionally seen, the vaginal polyp. This may show its presence by vaginal hemorrhage. The malignant growths have a tendency to increase in size and to invade neighboring tissues.

The vulva may occasionally exhibit acuminated warty growths of a benign epithelial nature (Fig. 323).

DISEASES OF THE UTERUS AND TUBES

The uterus may be entirely wanting or very rudimentary. Among other deformities of the organ are complete doubling of the uterus, and a partial division of the single uterus to varying degrees. One or both Fallopian tubes may be absent or rudimentary. Exceptionally inflammation of the uterus or tubes is found, sometimes of a gonorrheal nature, through extension from a gonorrheal vulvovaginitis. The use of a small speculum in such cases may show pus exuding from the cervix. In cases of imperforate hymen, the vagina and uterus may become dilated by mucous or watery secretion, and, if puberty has been reached, a hematocolpos or hematometra results. Uterine prolapse has been observed often combined with spina bifida. Complete prolapse has been reported even in the new born (Qvisling).[3] Malignant tumors of the uterus are very uncommon in children. Steffen[4] collected but 4 cases limited to the uterus and 6 affecting the vagina as well. Sarcoma is that most frequently found.

DISEASES OF THE OVARIES

One or both ovaries may be absent in very rare instances, or there may be accessory ovaries. **Ovarian hernia** is not rarely observed, especially in cases where the uterus and tubes are not fully developed. Puech[5] collected the reports of 78 cases. It is either congenital, or acquired in early infancy. The ovary on one or both sides may be found in the inguinal canal, less often beneath the femoral ring, and occasionally is displaced in other directions. Not infrequently in the case of inguinal prolapse the pedicle of the ovary becomes twisted, and pain, swelling and tenderness follow, and even strangulation occurs. Except for this accident, prolapse of the ovary usually produces no symptoms until puberty is reached. The diagnosis rests upon the position and shape of the organ, the dullness on percussion, the consistence, and the tenderness on pressure. If it is possible to restore the ovary to the normal position by manipulation, this should be done and a truss applied. Protection against injury must be used if the ovary cannot be replaced.

Morbid growths of the ovary are more frequent than those of the uterus, and are of various sorts; among them sarcoma, carcinoma, teratoma, adenoma, and cysts of different kinds. Mergelsberg[1] reckoned that at least 100 cases of sarcoma of the ovary in early life had been reported. They may be congenital, but are seen oftener in later childhood. Dermoid cysts and sarcoma appear to be among the most frequent varieties. The diagnosis from tumors of other organs is of great difficulty, and may, perhaps, not be possible before operation. The distinction is especially to be made from a morbid growth of the kidney. This latter is situated nearer to the flank, and often has the tympanitic resonance of the intestines in front of it. An enlarged spleen appears to start from under the left costal border. The only treatment is surgical, and is often successful, although the danger of operating is greater in early life than later.

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[1] De maligne Geschwülste im Kindersalter, 1905, 68.

[2] Inaug. Dissert., Berlin, 1913.

[3] Arch. f. Kinderh., 1890, XII, 81.

[4] Die maligne Geschwülste im Kindersalter, 1905.

[5] Gaz. obstet. de Paris, 1875, 129. Ref., Hennig in Gerhardt's Handb. der Kinderkr., 1878, IV, 3, 24.

TUBERCULOSIS OF THE FEMALE GENITAL ORGANS

This is generally secondary to the disease in other parts of the body, oftenest either the peritoneum or the intestines. It is much rarer than the analogous occurrence in the male. Brüning[2] reported on 44 collected cases in children. The external genitals (vulva, clitoris, labia) were affected alone in 2 cases, and in combination with the internal genital organs in 3. In 37 cases the process was confined to the vagina, ovaries, tubes and uterus; the last two being the most frequent seat. Gräfe[3] added 19 new instances, 10 of them occurring between the ages of 1 and 5 years, and 7 between 10 and 15 years. The condition is rather of pathological than clinical interest, as in most instances the diagnosis is a post-mortem one.

GENITAL HEMORRHAGE; PRECOCIOUS MENSTRUATION

The discharge of blood by way of the vagina may result from inflammation, injury, the presence of tumors of the vagina or asphyxia at birth; be an evidence of the hemorrhagic diathesis; or occur in various hemorrhagic forms of the infectious diseases, including sepsis. It is commoner in the new born than later, and was found by Zacharias[4] in 2.5 per cent. of 400 new-born female infants examined. The blood may arise from the vulva, vagina, or uterus. Genital hemorrhage may have occurred more than once without justifying the diagnosis of true **precocious menstruation.** This latter condition, in which a menstrual flow takes place at more or less regular intervals, and is often accompanied by other evidences of sexual and general percocity, is of great infrequency, although the interest attached to it is such that a number of cases have been reported. An interesting analytical review of various of the older instances is given by Hennig.[5] In the youngest of these patients menstruation was observed at the age of 2 days. A number of additional instances have since been published, and Lenz[6] has collected in all 130 cases. Some of the children menstruate for a time, and then cease; others continue regularly to and through adult menstrual life. Some age rapidly or become debilitated

[1] Dissert., Berlin, 1913.
[2] Monatsschr. f. Geburtsh. u. Gynäk., 1902, XVI, 144.
[3] Monatsschr. f. Geburtsh. u. Gynäk., 1914, XL, 448.
[4] Med. Klinik, 1914, X, 1643.
[5] Gerhardt's Handb. d. Kinderkr., 1878, IV, 3, 18.
[6] Arch. f. Gynäk., 1913, XCIX, 67.

and die early; in more no influence on the general health is apparent. The psychic condition is that characteristic of the age. A number have early become mothers. In a case reported by Dodd[1] the girl menstruated at 12 months, and gave birth to a child when slightly over 9 years of age. (See also Precocity, p. 538.)

Treatment of genital hemorrhage is seldom needed. Only occasionally is it profuse enough to necessitate local procedures. When the symptoms indicate some definite cause, this may demand therapeutic measures.

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[1] Lancet, 1881, I, 601.

SECTION VIII

DISEASES OF THE NERVOUS SYSTEM

Various classifications of the nervous disorders of children have been proposed, none of which can be entirely satisfactory. The fact that many diseases involve more than one region makes a division based on localization alone impossible, while on the other hand symptoms similar may be dependent upon diverse pathological causes. That employed here, as well as the order of arrangement of the various divisions, is adopted as merely provisional and a matter of convenience.

CHAPTER I

INDICATIONS OF SYMPTOMS

Anatomical and physiological peculiarities consequent upon incomplete development of the nervous system in early life, to which brief reference has already been made (Vol. I, p. 64) are the necessary causes of certain characteristics of nervous disease at this period. There is a consequent instability and excitability which renders the nervous system much more susceptible in many ways to pathological influences. The lack of complete development is observed in all parts, but is especially marked in the brain, and, as a result, convulsions, delirium, coma, and the like, are produced with great readiness. This depends upon the feeble inhibitory power of the nerve cells, the growth of which does not keep pace with that of the demands upon them. There is, further, imperfect functioning of those centres which control temperature, and it follows that children readily develop high fever from slight causes. The influence of inheritance is also very decided, as seen in numerous nervous disorders; and the great frequency in childhood of infectious diseases of many kinds is another factor rendering nervous disturbances very common. The prevalence and indication of some of the nervous symptoms seen in early life can receive attention here only to a limited extent. Especially the question of segmental diagnosis in its bearing upon paralysis and other symptoms is to be studied in special works upon nervous diseases in children, such as those of Sachs,[1] and of Bruns, Cramer, and Ziehen.[2]

Atrophy.—Many constitutional diseases are attended by widespread wasting, but localized atrophy is usually of nervous origin. Lack of use produces atrophy of the muscles to a certain degree, as in the cases of cerebral palsies, in which the paralyzed limb is smaller and shorter than normal; but the condition is most marked when either the anterior horns are involved, as in poliomyelitis and some other affections, or the nerves themselves. Here, in addition to the muscular wasting, there is glossiness, pallor, blueness, and coldness of the skin. In severe trophic disturbances bed-sores readily develop. Atrophy of the muscles is seen also to a high degree in the later stages of the various muscular dystrophies.

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[1] Nervous Diseases of Children.

[2] Handb. d. Nervenkr. im Kindersalter.

Paralysis.—Diseases such as rickets, syphilis, and scurvy may produce a condition of muscular weakness or of pain which simulates paralysis very closely, and a similar pseudo-paralysis may also be seen in hysterical conditions. True paralysis is dependent upon a number of causes, the lesions being located either in the brain, spinal cord, nerves, or muscles. Paralysis of both arms is oftenest due to poliomyelitis, less frequently to some form of muscular dystrophy; that of one arm alone may be dependent upon the former affection or upon injury to the brachial plexus, as in obstetrical paralysis, or upon a very limited lesion in the brain. In the last condition the paralysis is of a spastic nature, while in the others it is flaccid. Hemiplegic paralysis is oftenest cerebral and consequently of a spastic type; that of both lower extremities, if spastic, is usually dependent upon spinal caries, cerebral spastic diplegia, or upon diseases involving the lateral tract of the cord. Flaccid paralysis of one or both lower extremities occurs in neuritis, poliomyelitis and muscular dystrophies.

Ataxic movements, in which the movements are incoördinated, are normally present in young infants. Later they are oftener symptomatic of hereditary ataxia, cerebellar disease, and neuritis.

Choreic movements are seen, of course, most typically in chorea. Some of the habit-tics have a certain resemblance; but movements most suggesting those of chorea are found among the post-paralytic symptoms of the cerebral palsies.

Tremor is not a common symptom in early life. It may be general or limited. It may be dependent upon a cerebral lesion, but I have seen unilateral tremor of hemiplegic distribution occur as a purely functional disturbance.[1]

Nystagmus may be either vertical or horizontal. It is a symptom seen in a variety of nervous conditions, among them diseases of the brain, Friedreich's ataxia, gyrospasm, hydrocephalus, and disseminated sclerosis. Generally it indicates some organic disturbance, but sometimes is dependent upon reflex causes. It is also a frequent attendant upon local diseases of the eye, or it may be congenital or inherited, or occur sometimes in the weakened state resulting from gastro-intestinal disturbances in infancy.

Reflexes.—The tendon reflexes, particularly the *knee-jerk* which is the most important, although less active in the new born are normally during the first 2 years more active than in adult life. Vas[2] examined 200 healthy children from the 1st week of life upward and found the knee-jerk present in 98.8 per cent. and the Achilles jerk in 85.7 per cent. Abolition of the patellar reflex is seen in palsy of the flaccid type, as in poliomyelitis and neuritis; as well as late in the course of the muscular dystrophies affecting the lower extremities. This reflex is absent also in Friedreich's ataxia, and often in severe attacks of disease not of a nervous nature; such as enteritis and especially pneumonia. Often, however, it is difficult or impossible to elicit it, although no disease is present to account for this. Exaggeration of the patellar reflex is observed in cerebral and meningeal diseases, in systemic affections involving the lateral tracts of the cord, and in the compression myelitis of Pott's disease. Since the exaggeration may be present in some functional disorders also, the coexistence of *ankle-clonus* is a valuable corroborative evidence that the disease is organic.

[1] Transac. Amer. Ped. Soc., 1897, IX, 158.

[2] Jahrb. f. Kinderh., 1914, LXXX, 423.

The *Babinski reflex* (dorsal flexion of the great toe on stroking the sole) is of no value up to the age of 3 years, since it is often then elicited under entirely normal conditions. After this period its presence is pathological, and it is seen under the same conditions as the increased patellar reflex. Other reflexes will be referred to under the separate topics with which they are especially connected. (See also p. 322.)

Muscular Rigidity.—In the muscles of the neck this may be due to rheumatism (torticollis) or dependent upon disease of the cervical spine. It is seen here, too, in various forms of meningitis, and often in the acute stage of poliomyelitis, while retraction of the head with rigidity of the muscles of the neck may accompany a marantic condition in young infants, or be an attendant upon acute otitis. Elsewhere in the body rigidity may be produced in a similar manner or may depend upon the existence of a cerebral paralysis or of tetany.

Electrical Reactions.—These conditions have been clearly explained by Sachs in his text-book, to which reference has already been made. Normal electrical reactions are seen in all cerebral diseases, except those of the cranial nerve-nuclei; in disease of the lateral and posterior columns; in functional disorders; and in some muscular dystrophies. There is reaction of degeneration in poliomyelitis; neuritis; to some extent in Pott's disease; and, in general, in affections involving the anterior horns. Some of the muscular dystrophies show loss of or diminution of both faradic and galvanic response without reaction of degeneration, and the same is true of amyotonia congenita. Tetany gives an electrical response peculiar to itself. (See Tetany, p. 250.)

Sensation is diminished or lost, or there may be paresthesia, in neuritis. There is anesthesia also in any affection which involves the posterior nerve roots or the grey matter of the posterior horns. Consequently this symptom is present in transverse myelitis from spinal caries or other cause. Hyperesthesia is seen in many cases of irritability or inflammation of the nervous system; as sometimes early in neuritis, in poliomyelitis, meningitis, hysteria, and other conditions. Pain occurs in a great variety of disorders, as in neuritis, the various neuralgias, meningitis, brain-tumors, etc.

Cerebrospinal Fluid.—Of recent years the examination of the cerebrospinal fluid has come into constantly greater prominence in the study of nervous diseases. In normal children no fluid at all is obtained, or it is entirely clear; contains usually not more than at most 10 leucocytes to the c. mm., and is under a pressure of from 20 to 25 mm. (0.79 to 0.98 inch) of mercury, if the child is sitting, or from about 6 to 10 mm. (0.24 to 0.39 inch) if lying down. It contains sugar, as shown by the reduction of Fehling's solution; and both albumin and globulin, but the latter in traces only. When any form of meningitis develops, the reaction for globulin is positive, Lange's colloidal gold reaction is present, the cell-count and the pressure are usually above normal, sugar is often diminished, and in most instances bacteria may be found. The number and variety of the white cells varies. In purulent meningitis of any sort, the number is very greatly increased, and the majority are of the polymorphonuclear type. In tuberculous meningitis the fluid is clear or only slightly turbid, very decidedly increased in amount and the pressure often high. There is a distinct fibrin-coagulum, and the cell-count is generally from 100 to 200 to the c. mm., or often decidedly above this, the lymphocytes usually predominating eventually, although early in the case the neutrophiles may outnumber them. In serous meningitis the number of cells is still smaller, but is slightly above normal, the pressure is increased, and bacteria are absent or occur very sparsely. The fluid of poliomyelitis very closely resembles that of tuberculous meningitis.

Lumbar Puncture.—For the obtaining of the fluid lumbar puncture is required. The procedure is simple and easily done. The skin of the back should be well cleansed with soap and water and then with alcohol, and painted with iodine, the hands of the operator sterilized, or encased in rubber gloves, and the needle, preferably one of the stouter, longer ones supplied for exploratory purposes, thoroughly boiled. In place of a needle a small trocar and canula may be employed. The child either sits with the body bent forward and firmly held in this position (Fig. 324) or, I think preferably, lies on its right side, turned partially upon the abdomen with a small pillow beneath this. The knees and shoulders are then drawn well forward and firmly held by an assistant. In this position the spines and laminæ of the vertebræ become more widely separated from each other. An imaginary transverse line at the level of the crests of the ilia crosses the spinal column at the 4th lumbar vertebra. The needle, unattached to the syringe, is now introduced either in the 3d or the 4th interspace. It should be inserted in the median line and in a direction slightly upward. At a depth varying from 1 to 2 inches (2.5 to 5 cm.) it enters the subarachnoid space below the termination of the spinal cord. As a rule the fluid begins to drop at once, or sometimes, if the pressure is high, even to spurt. If it is purulent in nature, it may be necessary to attach the syringe, and to use slight suction to start the flow; but after this the syringe should be removed and the exudate allowed to escape unaided. Sometimes raising the head and shoulders slightly will suffice. The fluid should be received in a sterilized test-tube to allow of making cultures. A manometer devised for the purpose may be serviceably employed to measure the degree of intraspinal pressure present. At times a dry tap results, even in cases of undoubted meningitis. This may be due to the failure of the needle to enter the canal, to its being plugged with fibrin, to the thick character of the fluid, or to the fact that the inflammatory process has shut off the brain and the upper portion of the cord from the parts below. If the needle is introduced too far it will impinge on the opposite wall of the spinal canal. This accident does no harm except the wounding of small blood-vessels, as a result of which the

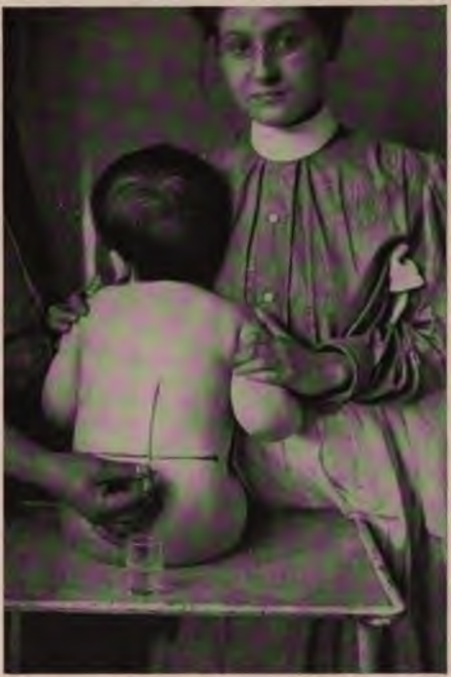

FIG. 324.—LUMBAR PUNCTURE PERFORMED WITH THE CHILD IN A SITTING POSITION.
Courtesy of Dr. Harry Lowenburg.

fluid which appears is at first bloody, and may continue to be so if there has been much hemorrhage.

The child must be prevented from sudden movement and the needle held loosely to avoid the danger of breaking it in the spinal canal—an accident which I have seen happen through neglect of these precautions and when too slender a needle had been employed. As a rule no anesthetic is necessary since the pain is but momentary and no greater than is produced by using an exploring needle elsewhere. If the child struggles violently against being held, and is weak enough to make this an objection, a few whiffs of ether may be needed, although this is generally as objectionable to the patient as is the slight pain of the puncture. The same remark applies to local anesthesia by freezing. Lumbar puncture is generally entirely without danger. The pulse should, however, be watched carefully, and no effort made to withdraw all the fluid possible, lest the degree of intracranial pressure be too violently disturbed.

CHAPTER II

FUNCTIONAL NERVOUS DISEASES

ECLAMPSIA

(Convulsions)

The term "convulsion" is often employed in a broad sense to designate any tonic or clonic spasm, with or without loss of consciousness, and general or local in distribution. It thus includes such manifestations of spasmophilia (p. 249) as tetany and laryngospasm, to be considered later; epilepsy; the so-called "inward-spasms;" the attacks of holding the breath; hysterical convulsions; paramyoclonus; habit-spasm; and other conditions. In the present connection, however, the title is applied only to the attacks of more or less general convulsions with loss of consciousness, which in early life are called "Eclampsia." This is perhaps to be regarded as a symptom rather than as an independent disorder, since it occurs in the course of a wide variety of diseases.

Pathogenesis.—An eclamptic convulsion is an involuntary irregular discharge of motor impulses from the cells in the brain, resulting in muscular contractions. The imperfect development of the inhibitory control of the cerebral nerve cells in early life, with the increased reflex irritability of the lower centres seen at this period (Soltmann)[1] readily allows the disease to develop. The tendency to convulsions is, of course, magnified by any condition which increases the irritability or disturbs the inhibitory control. With such existing, any slight stimulus applied either directly to the motor area of the brain, or reflexly from elsewhere in the body, may be a sufficient causative agent. It is evident, however, that the lack of stability of the nervous system is not of itself sufficient to occasion eclampsia; otherwise every infant would suffer. The theories of the exact mechanism are at variance. The experiments of Küssmaul and Tenner[2] showed that spasms followed the production of anemia of the brain; but certainly in many cases there is found at autopsy an intense hyperemia, possibly of later origin. Other causes, such as mechanical irritation of the floor of the fourth ventricle, may bring on convulsions, and it is very probable that toxic substances of various sorts may act directly as irritating agents.

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[1] Jahrb. f. Kinderh., 1876, IX, 106; 1877, XI, 101; 1878, XII, 1.

[2] Epileptiform Convulsions from Hemorrhage, 1859, New Sydenham Soc. Transl.

There are no lesions characteristic of eclampsia. Those found are extremely variable, and depend chiefly upon the causative agent.

Etiology.—The frequency of eclampsia is very great. In the mortality statistics it stands well toward the head of the list among the causes of death under 2 years of age. (See Vol. I, p. 214.) Age is an important predisposing factor, the great majority of cases occurring in the first 2 years of life, and especially in the 1st year. Of 7508 deaths accompanied by convulsions, collected by Lewis,[1] 4993 were in the 1st year, 1335 in the 2d, 893 were from 2 to 5 years, and 178 from 5 to 10 years. After the period of early childhood convulsions are seen much less often, except as evidence of some organic disease of the brain. The very large number of attacks of convulsions in infancy which do not have a fatal issue would increase the incidence greatly for that period of life. Sex appears to have little influence. Of 250 cases of convulsions studied by McIlraith[2] 52.8 per cent. were male and 47.2 per cent. female. Inheritance seems to exert some influence, several children of one family showing a tendency to the disorder, or the parents being of a distinctly neurotic type. Rickets is either itself a strongly predisposing cause or is a condition developing from the same nutritional disturbance which creates the liability to eclampsia. Whatever the truth may be, it is certain that rachitic subjects are especially prone to the development of the disease. Other disorders exhibiting nutritional disturbances likewise predispose, such as anemia, syphilis, lymphatism, malnutrition from any source, and spasmophilia; the subjects of the latter condition having a peculiar tendency to convulsions, often combined with laryngospasm or tetany. In fact eclampsia may in most instances be regarded as a manifestation of spasmophilia.

The **exciting causes** are of a great variety. They may be divided into several classes, which, however, overlap. Among them are the following:

1. The *acute infectious diseases* are frequently ushered in by a convulsion instead of the chill which would be observed in older subjects. This is especially true of scarlet fever and pneumonia; but the occurrence is seen frequently, too, in measles, typhoid fever and malaria. The exciting cause here is probably the direct first influence of the poison peculiar to the disease. Convulsions occurring later in the course of infectious disorders depend upon various causes. Thus decided hyperpyrexia may be a factor in some instances; in others toxic influences. In scarlet fever uremia fills an important etiological place; and in pertussis, in which eclampsia is especially liable to occur, the causes are diverse, such as asphyxia, cerebral hemorrhage, and exhaustion.
2. *Acute* and *chronic gastrointestinal disorders* constitute one of the most frequent causes of eclampsia, the poison produced in the course of the autointoxication probably acting directly upon the brain-cells.
3. *Toxemic* convulsions of other origin are seen in asphyxia, especially in the new born; uremia; and occasionally as a result of some of the metallic or vegetable poisons such as lead, opium, strychnine and the like.
4. *Hyperpyrexia* is an occasional cause. Although, as a rule, children bear moderately high temperature well, convulsions are liable to develop when this becomes excessive, as, for instance, in heat-stroke.

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[1] Keating, Cyclop. Dis. Child., 1890, IV, 862.

[2] Manch. Med. Chron., 1906, XLV, 80.

5. *Reflex irritation* may be of the most varied sort, and is one of the most frequent factors. The presence of undigested food in the stomach or intestines may bring about a convulsion by mechanical irritation before there has been time for toxic material to have been produced. Among other reflex causes are colic; earache; phimosis; trauma of any sort; retention of urine; burns; violent crying; fright; etc. Yet although the influence of many reflex irritations is of moment, it is only so if the susceptibility and predisposition of the subject is great. In fact, many conditions to which importance is often attributed, such, for instance, as worms, dentition, and the like, probably possess little, if any, influence under any circumstances.

6. *Disease or injury of the brain* is frequently accompanied by eclampsia. This is seen in meningitis of various forms; poliomyelitis; hydrocephalus; tumors; hemorrhage; embolism; and thrombosis. The sudden anemia following severe loss of blood, and the venous stasis accompanying diseases of the heart and lungs, may become etiological factors by interfering with the cerebral circulation. The majority of instances of convulsions in the new born probably depend upon intracranial hemorrhage. Strictly speaking these are in reality a form of symptomatic epilepsy.

7. *Hereditary syphilis* is liable to be attended by fatal convulsions in the new born, without any causative organic lesion being discoverable at autopsy.

8. *Terminal convulsions* are of frequent occurrence in infants dying from any disorder in which exhaustion is prominent. The development of eclampsia under these circumstances is in reality only a method of dying.

Symptoms.—Attacks of eclampsia vary much one from the other. They may be general or partial. In a typical, widespread, general convulsion there may be such premonitory symptoms as twitching of the mouth or extremities; flexing of the thumbs into the palms; rolling upward or crossing of the eyes; and restlessness, fretfulness or peevishness; but oftener the onset is sudden. There is sometimes an initial cry; the body stiffens; the elbows are flexed; the face becomes pale; the eyes have a fixed staring expression, or may be rolled upward; the pupils are contracted; respiration is momentarily arrested; consciousness clearly lost; and there is more or less general rigidity. In a moment the tonic spasm is succeeded by violent rhythmic clonic to-and-fro movements of the extremities, the face being distorted, cyanotic, and covered with sweat; the mouth often foaming; the lips cyanosed; the eyes congested; the pupils dilated; the head retracted or turned in various directions; the back arched; the abdomen flattened; respiration irregular and shallow. There is rattling of mucus in the throat; the pulse is irregular, rapid and weak; the hands made into fists with the thumb in the palm; the rectum and bladder may be emptied. The jerking movements of the body continue sometimes equally everywhere, sometimes now worse in one region and then in another. After a variable time—usually several minutes, sometimes longer—the convulsive movements grow less violent and frequent, respiration becomes regular, and cyanosis disappears; but the patient remains in a stuporous condition, frequently with a certain degree of rigidity persisting for a short time. Then complete relaxation follows, often with decided prostration if the convulsion has been severe, and the child passes into natural sleep or begins to cry.

Not infrequently, either after relaxation or without the rigidity having entirely disappeared, another paroxysm follows after some minutes or hours. In bad cases repeated convulsions may occur in this way during several days, with only short intervals of complete freedom; or with the partial rigidity and stuporous state continuing during the interval. Oftener, however, the convulsion is single.

Not all the symptoms as described are witnessed in every case of eclampsia. Often the convulsion is merely partial. It may be of only momentary duration, limited to a slight twitching of the extremities with brief cessation of respiration, crossing or rolling of the eyes, transitory slight distortion of the face, and unconsciousness. To this condition the laity often apply the term "inward-spasms," or "internal convulsions." In other cases the spasm begins upon one side, and either is confined here or spreads to the rest of the body; while in others there is little more than temporary loss of consciousness, and the symptoms simulate syncope. In still other cases the convulsion is so severe that cyanosis becomes extreme, respiration greatly interfered with, the extremities cold, the body covered with cold sweat, and the pulse too rapid and weak to be counted.

Prognosis.—This depends entirely upon the cause and upon the age. In the large majority of cases eclampsia is not fatal, unless the cause is an irremediable one. It is to be noted, however, that a child who has had one attack of convulsions is very liable to have others at some time. Death may occur in the first attack, but this is not common unless the patient is very young, or the convulsion unusually prolonged or severe. The danger is in proportion to the youth of the patient; the duration of the attack; the amount of prostration already present or produced by it; the degree of interference with respiration; the feebleness of the cardiac action; the intensity of the cyanosis and other evidences of asphyxia; and the depth of the coma. Convulsions frequently repeated are also of serious import. Asphyxia is the immediate cause of death in the short, very severe attacks; exhaustion in the long-continued and very frequently repeated ones. Convulsions occurring at the onset of an acute infectious disease are seldom of moment. If developing later, they are of more consequence. Spasms accompanying pertussis are always dangerous, as are those attending rickets or other debilitating conditions, or dependent upon uremia or upon asphyxia from any cause. Those due to reflex irritation are usually not dangerous. All convulsions the result of intracranial disease are of serious import, not so much in themselves, since they are more liable to be very frequent than very severe, but on account of the nature of the cause of which they are a symptom. It is on this account, as well as due to the early age of the patient, that convulsions in the new born are so often fatal. Those depending upon gastrointestinal disturbances are usually amenable to treatment, and their return can be prevented by appropriate measures. Those always beginning in or limited to one extremity give an unfavorable prognosis, since they point to a cerebral lesion. Convulsions which are first seen in early childhood may possibly be epileptic in nature. This is especially true if they soon recur and no cause whatever can be discovered for them. In general, the prognosis in any case of eclampsia, though as a rule good, must always be a guarded one. Not only is the recovery at the time uncertain, but the ultimate condition as well. The actual death-rate from convulsions is impossible to estimate, inasmuch as statistics vary so widely. This depends largely upon the fact that many patients are reported as dying of "convulsions," in whom these were only a terminal symptom, and the primary disease was the cause of the fatal issue.

Sequels.—Ordinary eclampsia leaves nothing in its train. Sometimes after severe attacks there is a temporary paralysis, the result of the exhausted nervous condition; or there may be aphasia, or a transitory stuporous state. These are all functional disturbances. Any other sequels of a permanent nature depend, usually, upon the condition of which the convulsion was a symptom. An exception, however, is the possibility of a convulsion producing cerebral hyperemia sufficiently severe to result in hemorrhage, with resulting permanent damage. There is danger, too, of convulsions which are frequently repeated over a long period leaving psychical disturbances as a sequel.

Diagnosis.—The recognition of a typical attack of eclampsia is easy. It rests upon the characteristic spasmodic movements and attendant symptoms, combined with unconsciousness. Some of the imperfectly developed seizures, however, may readily simulate syncope, attacks of holding the breath, or laryngospasm. Most important is the diagnosis of the cause of the occurrence, and to accomplish this a very careful study of the history of the patient and of all the symptoms present is required. Convulsions in the first few days of life are oftenest due to traumatic hemorrhage, or to asphyxia from atelectasis. In the latter case they are attended by cyanosis and feeble respiration, and in the former they are liable to be frequently repeated during, perhaps, several days. It is possible, too, for asphyxia to produce eclampsia after this period, as in bad cases of pneumonia, pertussis, cardiac disease, and diphtheritic laryngitis. In such cases the cyanosis and other symptoms indicate the nature of the cause. Convulsions dependent upon cerebral abscess or tumor are often partial in nature, or begin always in the same part of the body and then become general. It is possible, however, for limited convulsions to occur not dependent upon organic cerebral lesions. Imperfect cerebral development is a cause of attacks being repeated during a long period. There is no fever attending eclampsia due to cerebral disease of the kind mentioned; consequently the onset of general convulsions with high fever indicates the beginning of some acute infectious disorder, acute intoxication from digestive disturbance, or some form of meningitis. Only observation for a short time can determine the exact nature of the cause. In most of these conditions the attack is prone to be single; in meningitis there is liable to be frequent recurrence with the development of other evidences of this malady. Convulsions from reflex irritations or from spasmophilia are general and unattended by fever, unless the cause is one in which the temperature is increased by it. The influence of uremia in producing convulsions must never be forgotten, and an examination of the urine should always be made. I have seen convulsions in infants, apparently digestive in origin, shown to be uremic only by such examination. It is important to remember, however, that albuminuria is frequently found attending eclampsia from many causes, and does not necessarily indicate uremia. The powerfully predisposing influence of rickets, too, is to be borne in mind. As a general rule the convulsions of the new born are usually from asphyxia or cerebral conditions; and those in the first 2 years of life after this period are oftenest digestive. To this statement there are, however, so many exceptions that a careful study is required of each case.

Finally, the diagnosis of eclampsia from epilepsy is of great importance. In infancy idiopathic epilepsy is not probable, and the disease does not begin until after the age of 3 years in the majority of instances. At this early period a positive diagnosis is consequently hardly possible.

Later in childhood epilepsy is probable if no cause is discoverable; the attacks are repeated; there is aura, fall, or sudden cry; a stuporous sleep after an attack; and no fever. The diagnosis is often one of great difficulty, and only time can solve the question in many cases. There is no essential difference between symptomatic epilepsy and the convulsions in infancy dependent upon intracranial lesions. The terms are practically synonymous.

Treatment.—*Preventive treatment* is of the utmost importance in children who are evidently predisposed, or in those who have had one or more attacks. The cause is to be determined if possible, and treatment directed correspondingly. Especial attention should be given to the diet, since some fault here is so often the etiological factor. Whatever element of food is found to produce symptoms of indigestion must be eliminated. The child must not be allowed to over-distend its stomach with food of any sort. Rachitis must be brought under control as quickly as possible, and undue sensitiveness of the nervous system relieved. Over-excitement and fatigue of any sort are to be avoided, and the method of life changed in whatever way will most conduce to the improvement and maintenance of the general health.

For the treatment of the attack itself the first indication is its immediate relief by general measures. If the nature of the cause can be discovered promptly, the special procedures demanded by it are to be employed also. The child should be placed in a warm bath of 98° to 100° F. (36.7° to 37.8° C.) while a cold compress, frequently changed, or an ice-bag is applied to the head. The object of the bath is to produce relaxation. There is generally no need to add mustard to the bath, although this may be done if the effect of counterirritation is desired. An exception to this plan of treatment is found in the cases of great hyperpyrexia with a temperature of 107° or 108° F. (41.7° or 42.2° C.) or more. Here cold baths are indicated until the temperature is reduced to about 101° F. (38.3° C.)

As a rule the warm bath is sufficient to relieve the attack, but should this not be the case, the child should be removed from it and an inhalation of ether or chloroform given. At the same time there should be administered by enema a combination of chloral and potassium bromide, giving 1 or 2 grains (0.065 or 0.13) of the former, and 10 grains (0.65) of the latter to an infant of 6 months of age. The enema should be small, the buttocks should be elevated, and the solution be made to remain by pressing the thumb over the patient's anus. If the paroxysms are being repeated at short intervals, the same medication is preferably given by the mouth between the attacks, using a dosage half as great. By either method the dose may if necessary be repeated in an hour or more. For cases in which the convulsions recur as soon as the influence of the anesthetic has passed, nothing, in my experience, is better than a hypodermic injection of morphine, giving $\frac{1}{100}$ to $\frac{1}{50}$ of a grain (0.0006 to 0.0013) to an infant of 1 year, and repeating it in 2 hours if imperative. Among other remedial procedures recommended are the inhalation of nitrite of amyl, a brief compression of the carotids, lumbar puncture and inhalations of oxygen if asphyxia is decided.

In addition to these general measures, remedies directed to the cause are to be sought. Should there be evidence that the stomach is overloaded by a recent indigestible meal, an emetic of ipecacuanha may do much good, or lavage may be employed. If the food has entered the intestine, a large intestinal douche should be given, as well as a purgative, such as magnesia or castor oil, as soon as the child can swallow. All other sources of reflex irritation should be sought for and removed. Fever present may have been reduced by the warm bath. If not, and it is high, a cold pack can be given, or antipyrine in small doses. Uremia requires the treatment appropriate to it. Should any localizing symptoms be present in convulsions occurring in the new born, the existence of a meningeal hemorrhage is probable, and relief may follow operative measures for the removal of the clot. Generally, however, there are no localizing symptoms present which make operation possible.

As soon as the attack is over, measures must be adopted to prevent any early return. The intestines should be washed out, and a purgative given if this has not already been done. Care must be taken, however, not to employ procedures which distress the child greatly and cause excitement, lest a return of the convulsions be brought about rather than prevented. Thus should the child have just passed through an attack and be sleeping, it should not be disturbed immediately for treatment. During convalescence chloral and the bromides should be continued in smaller doses for several days; the child kept as quiet as possible; and the diet be of the lightest and most digestible, given frequently in small amounts. Later the measures referred to under preventive treatment are to be pursued.

EPILEPSY

In the strict sense epilepsy is a chronic convulsive disorder with loss or disturbance of consciousness, and is not dependent upon any known characteristic pathological lesion. To this the title *idiopathic* or *genuine* epilepsy is properly applied. There occur also epileptiform convulsions associated with organic cerebral lesions; and for these the title *symptomatic, organic* or *secondary* epilepsy is fittingly used. Although the latter would be more appropriately described under Organic Diseases of the Nervous System, the clinical manifestations of the two varieties are often so similar that for the sake of convenience they will be considered together. It must be said of these symptomatic cases that there must necessarily be present some predisposition in addition to the cerebral disease. Otherwise, all children with the same lesions would uniformly become epileptic; and this is not the case.

Pathological Anatomy.—As already stated there are no characteristic lesions present in idiopathic epilepsy. In cases dying in severe attacks there may be found various changes, such as edema and hemorrhages in the pia, the brain-tissue, the skin, or the internal organs; with recent parenchymatous alterations in the liver and kidneys. When the disease has been long-continued, increase of the glia of the cerebral cortex has been described by a number of investigators. In the cases of symptomatic epilepsy associated with organic intracranial changes, the lesions may be very varied, either localized or diffuse, congenital or acquired, gross or only discovered by microscopical study.

Pathogenesis.—The mode of production of a convulsion in epilepsy, and its nature, are akin to the conditions found in eclampsia. There exists some disturbance of the brain which allows the irregular discharge of impulses from the motor cells of the cortex. The disturbance differs from those producing eclampsia in that it is chronic and persisting. Its nature is unknown, but that it does not depend in any way upon physiological slowness of development of the inhibitory control, such as characterizes eclampsia, is shown by the tendency for epilepsy first to show itself at a period when the physiological lack of inhibition and the nervous over-excitability are diminishing. The motor cells of the cortex do not seem able of themselves to institute an epileptic attack, but need the action upon them of some stimulus, either existing in the brain itself or reflex from some other part of the body. The influence of reflex causes is very decided, as is that of various injuries to the brain; but always the epileptic tendency itself must be present.

Etiology.—The influence of *heredity* is very decided. There is a history of antecedent nervous disease or a neuropathic condition in nearly 50 per cent. of the cases. This applies to both the symptomatic and the idiopathic forms. Quite frequently children with idiopathic epilepsy are descended from parents or grandparents who had the disease. In 2400 cases analyzed by Gowers[1] 973, or 40 per cent., presented evidences of a neurotic inheritance, and in nearly three-quarters of these a history of antecedent or familial epilepsy was obtained. Of 553 children of 136 families, in which one parent had epilepsy, as reported by Echeverria[2] 78 suffered from epilepsy, 27 from other nervous diseases, and of the remainder 195 died very early from convulsions. In 132 cases tabulated by Finckh[3] epilepsy was present in one of the parents in 19 per cent. Figures at variance with these are those of Fairbanks[4] who in 175 epileptic children found a history of any convulsive condition in other members of the family in only 12 instances. Intemperance in the parents is another often quoted predisposing cause, as is syphilis, either by the producing of focal lesions, or by its general influence upon the nervous system. Epilepsy attacks both *sexes,* and the sex-incidence is disputed. In Gowers' series[5] 52 per cent. were females and 48 per cent. males; while in Lange's[6] 741 collected cases, 44.1 per cent. were females and 55.9 per cent. males.

As regards the relationship of *age,* idiopathic epilepsy is, with few exceptions, not a disease of infancy. It begins to be more frequent after this period is passed, and the great majority of cases first show the symptoms in later childhood or at puberty, the tendency having been present but dormant from birth. In 3002 cases analyzed by Gowers[7] the age incidence was: Under 10 years, 826; from 10 to 19 years, 1398; from 20 to 29 years, 463. After this period the incidence diminished greatly. The cases of epilepsy developing quite early are often associated with organic cerebral lesions. These may be the result of traumatism occurring before birth or later; or of hydrocephalus, tumor, a former meningitis, polioencephalitis, and the like. The symptoms of the disease may come on soon after birth, but generally not for several years. The predisposing influence of *eclampsia* occurring in early life is a much mooted question. It is probable that some of the cases of epilepsy apparently developing later in those who had suffered earlier from eclampsia were in reality epilepsy from the beginning. The tendency to the disease may have ceased for some years, to reappear as puberty approached. In other instances the occurrence of eclampsia in infancy and of epilepsy later is merely a coincidence, since certainly the great majority of cases of the former do not develop epilepsy. The influence of eclampsia may be said to be slight, if it exists at all, especially if we omit the cases of early convulsions dependent upon organic intracranial lesions.

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[1] Epilepsy and other Chronic Convulsive Diseases, 1901, 5.

[2] On Epilepsy, 1870. Ref., Binswanger, Nothnagel's Handb. der spec. Pathol. und Therap., 1904, XII, 1, 1, 77.

[3] Arch. f. Psychiat. und Nervenheilk., 1905, XXXIX, 825.

[4] Boston Med. and Surg. Jour., 1914, CLXX, 521.

[5] *Loc. cit.,* 2.

[6] Psychiat. Wochenschr., 1899, I, 320.

[7] *Loc. cit.,* 13.

The *direct causes* of an epileptic attack are various. Different reflex and toxic influences are operative, among those mentioned being fright, shock, or other psychic disturbance; diseases of the ear and nose; trauma; the occurrence of acute infectious diseases; masturbation; and especially digestive disorders. The action of most of these is slight unless the predisposition is great. Digestive disturbances, however, form an exception, the influence of these being certainly very decided. Yet the majority of epileptic convulsions can be traced to no immediate cause whatever.

Symptoms.—The typically developed attack of *idiopathic* epilepsy goes often under the name of *major epilepsy* or *grand mal.* In addition to this there exists a large variety of attacks of a modified, milder character, often rudimentary, and grouped together under the title of *minor epilepsy,* or *petit mal.* The symptoms of *symptomatic epilepsy* in many cases are of a nature which points to the existence of a focal cerebral lesion.

Major Epilepsy.—In the typical attack of "grand mal," there are in many cases prodromal symptoms of very short duration, known as the *aura.* This is varied in nature, being, in general, sensory, motor, vasomotor, or psychic. There may be a disturbed sensation of some sort in the pharynx, the region of the heart, the stomach, or some other part of the body; such as pain, numbness, or tingling. Motor auræ may be represented by slight twitching or paresis of some part of the body, as sometimes of the tongue producing temporary inability to speak; and vasomotor auræ by flushing or pallor of some region. There may be sudden feeling of dread or anxiety, restlessness, excitability, or other psychic condition of very varied nature, or disturbances connected with the auditory or optic apparatus, such as flashes of light, momentary blindness, vertigo, sounds as of whistles, bells, and the like. Some of the rare cases designated as *procursive epilepsy* are instances of an unusual aura in which there is a tendency to run rapidly and without purpose for some moments before falling in the ordinary fit. Whatever the aura may be, it is a warning to the child that an attack is about to begin. Its duration varies; generally but a few seconds, but exceptionally lasting even a half minute. In many cases of epilepsy there is no aura, and the onset is entirely sudden. The attack then begins with an abrupt loss of consciousness, pallor of the face, dilated pupils, and often a cry; and the child falls as though shot, if in a position to do so. Immediately there occurs a tonic spasm more or less widely spread throughout the body. The head is retracted or drawn to one side; the jaws are tightly shut; the face cyanotic; the extremities are rigid; the forearms flexed; the hands clenched; respiration is interfered with by the contraction of the muscles of the chest. This tonic spasm lasts a few seconds up to half a minute, and is then followed by the clonic stage with movements and symptoms similar to those of eclampsia. During this period the tongue is often bitten as a result of the spasmodic movements of the jaws, foam, often bloody, comes from the mouth; the face is distorted; the eyes roll in different directions; cyanosis persists, but to a less degree; there are irregular rhythmic spasmodic movements of the trunk and extremities; respiration is jerking and irregular; the bowels and bladder are often emptied involuntarily. This stage of the disease lasts a variable time, usually only 2 or 3 minutes, sometimes longer; the symptoms gradually growing less severe, then disappearing, and the patient, relaxed, passing into coma with snoring respiration from which he cannot be wakened. This in turn lasts from a few minutes up to a quarter of an hour, and the patient can then be roused or perhaps awakes of himself bewildered; yet oftener, if not disturbed, passes into a natural sleep. From the beginning of the attack there is complete loss of consciousness and insensibility to pain. A moderate elevation of temperature is frequent during the attack, and the urine not uncommonly contains albumin, rarely sugar. Some headache, fatigue, or general pain may be experienced when the child wakens from sleep.

Not all instances of major attacks show the symptoms so completely developed, or following this normal sequence. Thus occasionally the convulsive movements begin in some one region, and the consciousness is only fully lost as the spasm spreads to the rest of the body. This does not necessarily indicate the existence of a focal organic lesion.

Minor Epilepsy.—The frequency of minor epilepsy is greater than that of the fully developed form. There exist many diverse modifications, showing all degrees of intensity, connecting this with the major variety. In all there is the characteristic complete or partial loss of consciousness, although often momentary. The child may suddenly stop talking; have a far-away expression of face, with pallor; and then in a moment continue his conversation at the point of interruption without knowledge that anything has happened. Any object in the hand will probably be dropped. Thus very mild attacks may be supposed by the parents to be dizziness, absent-mindedness, faintness, and the like; and their true nature never suspected. The patient may in some cases have a sensation of dizziness or faintness, but there is seldom a distinct aura. Sometimes there is confusion of mind for a moment after the attack, and automatic acts of very varied sorts may be performed, such as partially undressing; and occasionally these acts may be violent. In other instances there may be a few slight twitching movements of the face or a trembling of the body accompanying the seizure. Still other cases are more severe in that the loss of consciousness is attended by staggering or by falling, although this loss is of such brief duration that with the violent contact with the floor consciousness returns, and crying begins on account of the pain from the fall. In some of the cases of procursive epilepsy, already referred to (p. 244), there is a disposition to run rapidly for a few moments, the consciousness being sometimes not entirely lost, and no general convulsion follows. These belong properly to the category of petit mal.

Symptomatic Epilepsy.—The symptoms of this form of epilepsy may be either general in type, or localized and perhaps hemiplegic; or may remain hemiplegic over a long period and then become general. When general in nature, the first movements in any attack are liable to be seen in the region presided over by the portion of the brain affected. They may, however, be widespread from the very beginning of the seizure and distinguished in no way from those of the idiopathic form. Any aura occurring is liable to be motor in character and may be an indication of the seat of the organic lesion. There is, as a rule, loss of consciousness; but in one form this is preserved either throughout or until late in the attack. This is the condition to which the title *Jacksonian epilepsy* has been applied.

Course of the Disease.—The beginning of epilepsy is often overlooked. Sometimes the only attacks are during the night (*nocturnal* *epilepsy; epilepsia larvata*), and it is only on account of the occurrence of bed-wetting on certain occasions, combined with injury to the tongue and with exhaustion or headache found present in the morning, that the disease is suspected. Generally the early attacks of idiopathic epilepsy occur at first at long intervals, perhaps 1 or 2 in a year; and then become more frequent, although usually separated by intervals of weeks or months. Sometimes the disease begins with well-marked attacks of grand mal; in other cases with petit mal, which gradually alters in character to that of major epilepsy; or there may be an alternation of an occasional major attack with a number of minor ones. There is always a tendency for petit mal to change gradually into the more severe form. The attacks of petit mal are usually more frequent than those of the major form, and sometimes reach as many as 20 or 30 up to occasionally nearly 100 in the course of the day.

In some severe cases of major epilepsy the seizure, instead of completely disappearing, is rapidly succeeded by others without recovery of consciousness (*status epilepticus*). The condition is a serious one but not common. It occurs oftenest in symptomatic epilepsy. In this form, too, the attacks are liable each to be long-continued, at least at first, and to occur frequently, and finally to assume the character of grand mal, if they have not had this from the beginning. In general, symptomatic epilepsy exhibits a more rapid increase in the frequency of the attacks than does idiopathic epilepsy; the status epilepticus is oftener seen; and the psychic degeneration is more frequently present and is greater.

The general health of most patients with epilepsy suffers but little from the disease, unless the seizures are very frequent and the course prolonged, in which event there may be gradual loss of strength. The mental condition, however, is usually not entirely normal. There may be merely a change of disposition or character, the children being excitable, irritable, or subject to attacks of violent temper; or they may be simply backward in intellectual development. All varieties of epilepsy are liable to exhibit psychic changes, which increase in significance under the influence of a long continuance of the disorder, and may even develop into imbecility. This is perhaps especially true of cases of frequently repeated petit mal, and of those secondary to organic cerebral disease. There are, however, many exceptions to these statements, particularly in major epilepsy, and some individuals are and continue to be entirely bright and normal in every respect between the attacks.

Prognosis.—Epilepsy is seldom directly fatal during an attack, except as a result of some accident, such as drowning from a fall into the water, falls upon the head or into contact with a flame, or as the result of exhaustion in the condition of status epilepticus. The prognosis as regards final recovery is, on the whole, unfavorable. Those cases associated with organic cerebral lesions seldom recover, and death is liable to occur from some intercurrent disease or gradual loss of strength. An exception is seen occasionally where a distinct focal lesion can be determined, which is of a nature susceptible of removal by operative treatment. Instances of idiopathic epilepsy in which there is a distinct inheritance give an unfavorable prognosis as regards ultimate cure, as do those in which the disease does not begin until well on in later childhood or after this period; and those also of earlier development which have continued several years without benefit by treatment. Frequency of the attacks makes the prognosis worse, as does the presence of psychic involvement.

The prognosis is best where some strongly acting influence is present and can be removed; such as disturbance of the digestive apparatus; anemia; syphilis, etc. Long-continued cases with frequent seizures are liable to develop mental weakness, or even imbecility, or to become much weakened in body, rendering the patient subject to death from complicating diseases, among them tuberculosis.

In general the prospect of an ultimate complete cure of epilepsy is slight. Often the occurrences cease for long periods when treatment is continued, only to return later. It is not uncommon, too, for attacks of epilepsy to be present in early childhood and then cease entirely, spontaneously or under treatment; but for the disease to return in force at puberty or later. All this makes caution in prognosis necessary, even in cases apparently doing well.

The duration of life is very variable. The severer cases associated with organic cerebral lesions and suffering from very frequent attacks are liable to die during childhood or before adult years are reached. The duration in idiopathic epilepsy depends largely upon the severity and frequency of the seizures. Many cases, as stated, show mental and bodily degeneration and die on this account. Others live for many years without much interference with the general health by the disease, and the mind may continue exceptionally bright. Of this there are numerous well-known instances in history. It is difficult to estimate the actual mortality in epilepsy. In a series of 2029 cases in institutions collected by Worcester,[1] 30.11 per cent. died.

Sequels.—A transitory paralysis or paresis may occur, hemiplegic or paraplegic, and the result of exhaustion; or a condition of aphasia may be a sequel after the status epilepticus. The changes which may gradually take place in the psychical condition of the patient in long-continued cases have already been referred to. The psychic defects especially common in symptomatic epilepsy are oftener the result of the lesion than of the epilepsy itself. Naturally, the repeated falling occurring during the prolonged course of the disease is liable to result in physical injury, as shown by the permanent scarring of the face often observed, and in other ways.

Diagnosis.—This is usually easy in typical cases of the major form. It rests upon the repeated development of seizures with sudden onset, cry, and fall, and the typical convulsive movements as described. A single attack of this nature is entirely inconclusive. It is only by observing the chronic character of the disease that a diagnosis can be made. When the paroxysms occur solely at night, the diagnosis must be based upon the combination of biting of the tongue and of bed-wetting, together with headache or exhaustion upon the following morning; unless by chance the family have observed the seizure. Minor epilepsy is so varied in its manifestations that only continued study, and the observation of the chronicity of the affection, may make its recognition possible. The absence of entire or partial loss of consciousness is against the diagnosis of epilepsy of this form.

There are a number of conditions from which epilepsy must be distinguished. The convulsion of *eclampsia* is almost exactly like that of major epilepsy, and an early diagnosis is often impossible. The earlier in life the convulsions occur, the more liable are they to be eclamptic. Eclampsia is a disease especially of the first 3 years of life; epilepsy, except the symptomatic form, is chiefly one of later childhood or puberty.

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[1] New York Med. Rec., 1888, XXXIII, 467.

In symptomatic epilepsy, too, some evidences of cerebral lesions other than convulsions are usually present. In eclampsia there is generally some readily discoverable cause to which the convulsions are secondary, the sudden fall is not so often seen, and the aura is absent. All this applies, however, in a general way; and in individual cases the passing of time alone and the careful study of the case can make the diagnosis certain. As has been stated elsewhere, eclampsia in infancy dependent upon intracranial lesions is in reality symptomatic epilepsy.

The differentiation between attacks of minor epilepsy and *syncope* is often difficult. The loss of consciousness and the consequent fall in syncope is, however, not so sudden, and there are no convulsive movements or other suggestive symptoms. The urine is not passed involuntarily; there is no biting of the tongue; the pupils are not dilated, and the pulse is feeble. The chronicity of petit mal will eventually resolve any doubt.

The diagnosis of epilepsy from *hysteria* is often of the greatest difficulty. The question does not arise as frequently in children as later in life. While falling is seen in hysteria the tongue is not bitten, injuries of other sorts do not occur, the bladder is not emptied, nor is there analgesia during the state of apparent unconsciousness. It is minor epilepsy, however, which causes the greatest perplexity. The manifestations of hysteria are as varied as are those of this disease, and careful study is required to distinguish between the two. In favor of hysteria is the remembering by the patient of the details of the attack. I recall an instance where only in this way was the existence of a procursive epilepsy disproved.

The diagnosis of *symptomatic* from *idiopathic* epilepsy is difficult when the former exhibits generalized convulsions. The past history of the patient; the fact that the attacks begin always in a certain region, or that at one time they were strictly localized; and the discovery of some trace of paralysis, increased reflexes, headache, or other symptoms of an old or recent cerebral lesion point toward an organic epilepsy.

Treatment.—During the seizure only such measures are required as will prevent injury. The patient should be placed in a comfortable position, the clothes loosened about the neck, and a cork or rubber placed between the teeth to prevent the biting of the tongue. If there is an aura, inhalation of nitrite of amyl will sometimes prevent or shorten the attack. There is nothing to be gained by the forcible holding of the patient, and the efforts to unclench his hands, so in vogue among the laity. In very severe or prolonged attacks, as in the status epilepticus, chloral, morphine, or inhalations of chloroform or ether may be used.

The treatment of the disease as a whole should be, first of all, general and dietetic, the procedures varying with the case. Excitement and over mental work must be prevented; and in fact the life should be largely out of doors, and as regular, quiet and hygienic as possible. In some instances rest in bed a portion of every day is beneficial. Children having frequent attacks should not be sent to the ordinary school, but instructed at home, or in bad cases in special institutions, both for their own sake and for that of others. Cases in which the mind is becoming decidedly involved are much better placed in an institution for epileptics.

The causes which bring on an attack must be diligently sought for, and receive appropriate treatment. Particular attention must be given to the digestive apparatus. In some cases a milk-diet is of benefit; in ot [illegible] limiting of the amount of meat is of value. In all cases the diet should be light and digestible. Tea, coffee, and alcoholic beverages must not be given. Constipation is to be avoided with especial care, and a laxative administered at regular intervals is advisable. When syphilis is suspected, the treatment proper for this should be instituted.

The principal medicamentous treatment consists in the administration of the bromide salts, or of some of the newer preparations of bromine. The dose should be of a size sufficient to prevent the attack if possible, but no greater. Relatively large doses are borne by children, but a severe bromide eruption is sometimes unexpectedly produced. (See Fig. 422, p. 549.) A child of 5 or 6 years of age may take for a time from 25 up to even 75 grains (1.6 to 4.8) of a bromide salt daily, well diluted with water. If the attacks occur oftenest at any fixed hour in the twenty-four, the main dose for the day should be given some hours before this. So, too, if a fairly regular interval of days or weeks passes between the seizures, relatively smaller amounts may be given in the intervals, increasing these very decidedly before the expected attack. If cessation or amelioration of the disease follows, the administration of the bromide must be continued in smaller doses, if possible for months or even years. Should decided bromism necessitate the stopping of the remedy for a time, or should the treatment fail to relieve, other drugs may be tried, although from them, as a rule, there is not as much to be expected. It has been claimed that the bromides are most efficacious if salt is withdrawn from the diet. Among the numerous other drugs recommended are chloral, antipyrine, acetanilid, arsenic, chloretone, digitalis, belladonna, borax, nitrate of silver and opium. I have in some cases had remarkable results with the alternation of opium and the bromides, according to the method described by Flechsig;[1] the former being given in increasing amount for some weeks, and then suddenly replaced by large doses of the latter.

Operative treatment has been often tried for cases of symptomatic epilepsy. The high hopes once entertained have not been fulfilled. Good results, however, sometimes follow; and surgical aid should always be invoked in selected severe cases of this sort not relieved by medicinal treatment; as where there is reason to believe that a small cortical lesion of any sort exists.

SPASMOPHILIA

(Spasmophilic Diathesis)

Under the title spasmophilia, as first used by Thiemich,[2] is designated the condition seen in children in which there is a peculiar tendency to convulsive disorders of various sorts, notably some of the instances of eclampsia, laryngospasm, and especially tetany. In fact, the term "tetanoid condition" has been employed as a synonym, although it does not cover all the manifestations. Many general convulsive seizures dependent solely upon autointoxication from digestive disturbances, or upon infections of some sort, are not to be included in this category.

The chief characteristic, apart from the actual spasmodic attack, is the increased mechanical and electrical excitability, to which fuller reference will be made in discussing tetany. This latent evidence of spasmophilia may exist in children who never suffer from the active convulsive manifestations of the disease. Probably the majority of cases of spasmophilia are latent, and only revealed by the electrical or mechanical tests. The disturbance is a functional one, based upon no known pathological lesion, and apparently not connected with the imperfect development of the nervous system characteristic of all children in early life. The active spasmodic symptoms are probably brought about oftenest by some reflex irritation. The disease in its latent form (*latent tetany*) is probably a rather common one, although with distinct manifestations, and especially in the form of tetany, it is much less often seen.

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[1] Neurol. Centralbl., 1893, XII, 229.

[2] Münch. med. Wochenschr., 1899, XLVI, 1449. Spasmophile Diathese, Finkelstein, Lehrb. der Säuglingskr., 1905, I, 238.

The **etiology** is not entirely clear. There is sometimes evidence of inheritance. Pincherle and Pollidori[1] found a familial factor present in 26 per cent. of 91 cases. The condition seems oftenest to begin within the first 2 years of life and to cease in the 3d or 4th year; but, exceptionally, no symptoms are seen until the 3d or even the 8th year. There is a distinct tendency for the attacks to develop in the winter and spring, as demonstrated by the statistics given by Escherich[2] in which, out of 246 cases, 216 occurred from November to April inclusive, the largest monthly number being 53 during March. The influence of the diet is very decided, as shown by the infrequency of the condition among breast-fed infants; but just what the active element is in producing the disease is uncertain. There is a clinical association of spasmophilia with rickets; yet it is uncertain whether rickets is itself the cause of spasmophilia, or whether both depend upon the same or allied underlying disturbed metabolic processes. The same uncertainty of relationship applies to the status lymphaticus (Vol. I, p. 632) and to the exudative diathesis (Vol. I, p. 630), each of which is frequently combined with spasmophilia. It is probable that all are allied in their origin, and are consequently liable to be encountered in the same individual. Congenital debility appears to exert some influence, Rosenstern[3] having found evidences of spasmophilia in 76 per cent. of cases of this condition. The nature, cause and relationships of spasmophilia are referred to further in discussing Tetany and Laryngospasm.

TETANY

(Arthrogryposis. Carpo-pedal Spasm)

According to Fischl[4] tetany was first described by Etmüller in 1708, and later the first epidemic was reported by Wolf in 1717. The earliest description of the disease in children was by John Clarke in 1815. The name "Tetany" was used to designate it by Corvisart.[5] "Arthrogryposis" is the title applied to the carpo-pedal spasms in which only the hands and feet are involved. By some this has been considered a distinct disease; but the two conditions shade into each other and are undoubtedly the same with varying manifestations.

Etiology.—This has been considered in part in discussing spasmophilia (p. 249). As far as children are concerned, the disease, omitting this latent form (see above) is not a common one, and is to a large extent confined to children up to 2 years of *age*. In Baginsky's statistics of 107 cases in children,[6] 97 were in the 1st year of life, and in those of Rilliet and Barthez[7] on 87 cases in the first 15 years, 36 were in the 2d year.

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[1] Riv. di clin. pediat., 1918, XVI, 169.

[2] Traité des mal. de l'enfance, Grancher, 1905, IV, 404.

[3] Zeitschr., f. Kinderh., Orig., 1913, VIII, 171.

[4] Deut. med. Wochenschr., 1897, XXIII, 150.

[5] De la Contracture d. extremit., ou tetanie chez d'adult, 1852. Ref. Soltmann, Gerhardt's Handb. d. Kinderkr., 1880, V, 1, 1, 141.

[6] Lehrb. d. Kinderkr., 1902, 598.

[7] Barthez et Sanné, Mal. des enf., 1884, I, 419.

In my own 77 collected cases in American literature up to 1895,[1] including 5 personal cases added, and not limited to childhood, 34 were over the age of puberty, and 25 in the first 2 years of life. I am convinced, however, that these figures do not represent the actual age-incidence. Howard[2] has added 77 additional collected American cases up to 1906, including 9 of his own; 30 in adults and 47 in children. Sex possesses some influence. In infancy and childhood males are oftenest attacked. At puberty and in adult life tetany is more liable to develop in females. The geographical distribution varies remarkably. In America tetany is less common than in some other regions, although undoubtedly very often overlooked. I have observed it many times since the publication referred to. In some European countries, on the other hand, it is quite frequent. It has appeared almost epidemically at times, and is always more common at certain seasons of the year, chiefly winter and early spring. In 105 cases collected by Frankl-Hochwart,[3] 107 occurred in the months from January to April inclusive.

Among other conditions influencing the development of tetany are especially *rachitis* and *gastrointestinal diseases*. It is beyond doubt that the great majority of cases of infantile tetany develop in rachitic subjects. Escherich[4] estimates that 95 per cent. of all the cases of tetany seen at the clinic in Graz are sufferers from rickets; and Kirchgässer[5] found rickets recorded in 79.85 per cent. of 283 collected cases of tetany. Whether rickets is the cause, or is the result of the same underlying disordered metabolic processes, is not certain. Gastrointestinal disturbances likewise are intimately associated with the production of tetany, the disease perhaps being brought about by the resulting toxemia. It seems certain in any event that the symptoms of tetany can be influenced by the character of the food given, and that it is especially liable to develop in children fed upon cow's milk; and there is evidence to show, as pointed out by Finkelstein[6] and others, that there is some element in cow's milk which is a powerful direct factor in producing the disease. Among other causes mentioned are exposure to cold, intestinal worms, various infectious diseases, the status lymphaticus with enlargement of the thymus gland, disease or removal of the parathyroid glands, and deficiency of calcium salts in the body. There is certainly (Vol. I, pp. 630, 632) an intimate relation between the status lymphaticus and evidences of spasmophilia; but whether or not, as with rickets, this is more than a dependence of both upon the same or allied causes is not certainly known. As regards the influence of the *parathyroid glands*, surgical experience as well as autopsies and animal experimentation show that the removal of these glands, or hemorrhagic or degenerative processes in them, may be followed by tetany. There is, however, abundant evidence upon the other side, to the effect that not all cases of tetany depend upon the parathyroids. So, too, the influence of a deficiency of *calcium salts* in the cortex of the brain (Quest)[7] is still undetermined in spite of the large amount of investigation upon the subject. Although there is reason to believe that the administration of calcium in full doses will often relieve the symptoms of tetany, I have seen just the reverse occur;

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[1] Amer. Jour. Med. Sci., 1895, Feb.

[2] Amer. Jour. Med. Sci., 1906, CXXXI, 301.

[3] Nothnagel's spec. Path. u. Therap., 1898, XI, 2, 116.

[4] Traité des mal. de l'enf., Grancher, 1905, IV, 420.

[5] Deut. Zeit. f. Nervenheilk., 1900, XVI, 356.

[6] Fortsch. d. Med., 1902, XX, 665.

[7] Jahrb. f. Kinderh., 1905, LXI, 114.

and Stölzner[1] concludes from his experience that tetany is in reality a calcium-intoxication. There is, nevertheless, increasing support of the view that a deficiency of calcium salts is capable of producing the disease, at least in some instances, and it is possible that the parathyroid glands normally exert some control upon the retention of calcium in the sytem, and that this action is absent in cases of tetany.

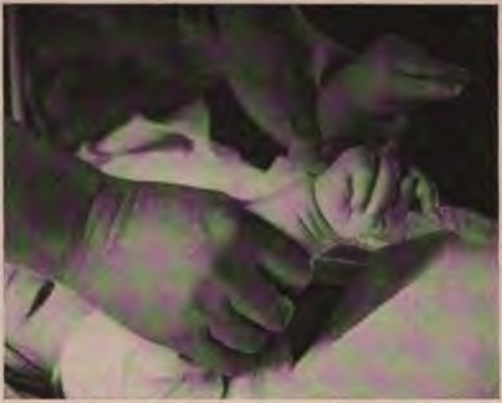

FIG. 325.—TETANY.

Male infant of 1½ years, in the Children's Hospital of Philadelphia. Somewhat rachitic. Illustration shows the flexion of the fingers and the position of the thumb often seen.

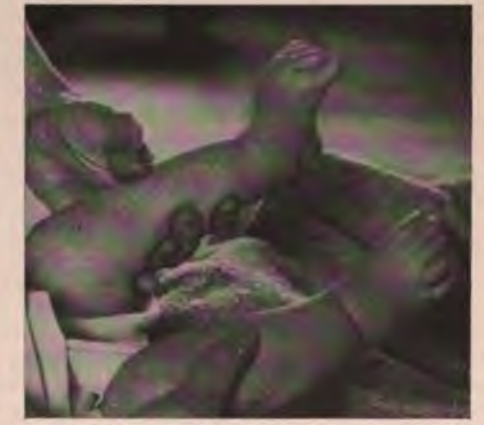

FIG. 326.—TETANY.

Same case as in Fig. 325. Well-marked plantar flexion of the toes.

Symptoms.—The onset is usually sudden. The symptoms as seen in early life consist oftenest of a tonic spasm in the hands. As a result the thumb is drawn into the palm, the fingers adducted and flexed at the metacarpo-phalangeal articulations, but extended at the more distal joints (Fig. 325), and the wrists flexed. There is thus produced the "accoucheur's hand," as it has been called. In other cases the fingers are tightly flexed over the thumb in the palm. Often, too, the forearm is flexed, and the upper arm drawn to the body. Usually the spasm soon extends to the lower limbs as well, in which event the foot is extended in the position of talipes equinus or equinovarus, the toes flexed on the sole, and the sole itself hollowed (Fig. 326). After lasting a short time decided edema develops on the dorsum of the feet and the back of the hands and wrists. Less often the knee-joints are rigidly flexed, and the thighs flexed and adducted. In most cases the contraction is limited to the extremities, especially the hands and feet. Occasionally it occurs in the feet only. Very rarely in infancy there is in addition to the contraction of the extremities a widespread tonic spasm throughout the body, including the muscles of the trunk and the face, and interfering with respiration and swallowing. This condition is more characteristic of tetany occurring in adult life. There is never any involvement of consciousness.

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[1] Jahrb. f. Kinderh., 1906, LXIII, 661.

With the tonic spasm there is often paresthesia together with pain, the latter causing crying and being made worse if passive movement of the affected muscles is attempted. Frequently, however, especially in cases which have lasted for some time, there is no pain unless the parts are disturbed, and the child seems comfortable and makes efforts to use the hands as far as possible. The tendon and cutaneous reflexes are exaggerated.

The two symptoms most diagnostic of tetany, yet not always obtainable, are those characteristic of spasmophilia in general. These are increased mechanical excitability of the nerves, and altered electrical reactions. The mechanical irritability is well seen in the facialis symptom, or Chvostek's[1] sign. A slight tapping or stroking in front of the auditory canal or over the malar bone causes sudden clonic contraction of the muscles of the face. The indications and the frequency of occurrence of this symptom is disputed. I have found it present in many instances, and have been unable to obtain it in other well-marked cases of tetany or of laryngismus stridulus. Hochsinger[2] thinks it always of pathological significance in older children, indicating the existence of a congenital neuropathic constitution. Raudnitz,[3] as a result of the examinations of 2024 individuals, found it rare in children under 5 years, but so common after this period that it could not be called a sign of disease. The conclusion may be drawn that, occurring after the period of infancy, it cannot be called a proof of the existence of tetany; while appearing in infancy it points very strongly to the existence of a spasmophilic state; although its absence is no proof that this does not exist. Another evidence of the same excitability is the Trousseau's[4] symptom. If, namely, firm circular pressure is made over the nerves and arteries of the upper arm, the tetanoid position of the hands will be increased. These symptoms are very characteristic of tetany and are present in many cases.

The altered electrical reaction of tetany described by Erb[5] shows an increased excitability of the nerves to the galvanic current, a current-strength of 5 milliamperes or less, which would produce no contraction on cathodal closing in a healthy child, causing this to appear, even tetanoid in type, in one with tetany or other evidences of spasmophilia. Still more characteristic is the obtaining of contraction with the cathodal opening with the same weak current, as well as the fact that an anodal opening contraction is produced with less current strength than on anodal closure (Thiemich).[1] The electrical reaction can be examined satisfactorily in the median or the peroneal nerve.

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[1] Wien. med. Presse, 1878, XIX, 822.
[2] Wien. klin. Wochenschr., 1911, XXIV, 1487.
[3] Deut. Gesellsch. f. Kinderh., 1913, XXX, 62.
[4] Journ. f. Kinderkr., 1851, XVII, 335.
[5] Ziemssen's Handb. d. spec. Path. u. Therap., XII, 2, 1, 335.

The studies of Holmes,[2] based upon the examination of a large number of healthy children, as well as of others with spasmophilia, are of importance in this connection. The conclusions were that a cathodal opening contraction under 5 milliamperes is pathognomonic of tetany in children under 5 years of age. An anodal opening contraction, obtained with a less current-strength than for the anodal closing contraction, and with less than 5 milliamperes for the first 6 months of life, and of 2 milliamperes from the age of 4 to 5 years, is probably diagnostic of tetany, but is of little value after this age.

This mechanical and electrical excitability is obtained not only when the symptoms of tetany are actively present, but between attacks or in subjects with the spasmophilic diasthesis who have never as yet shown pronounced symptoms (*latent tetany*). There are, however, exceptions, and I have failed at times to obtain the Erb reaction even when there was every reason to believe that spasmophilia existed.

There are two types of tetany; the *intermittent* and the *persistent*, with various transitional forms. In the typically intermittent cases, oftenest seen in adult life, the spasms may be widespread; last only a few minutes or an hour; are frequently repeated and painful; and the electrical and mechanical excitability are very marked. In the persistent form, more characteristic of infancy, the spasm is oftenest limited to certain regions, especially the hands and feet, and may be continued for hours, days or weeks; although in the last event the contractions may lessen or even cease for some hours or longer, and then be renewed. Pain is not a prominent symptom, and the increased mechanical and electrical excitability is often difficult to elicit (Escherich).[3]

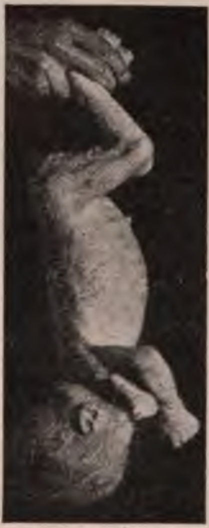

FIG. 327.—HYPERTONIA IN AN INFANT OF 11 WEEKS.

Had been fed on rice-water and condensed milk. The illustration shows the persistence of flexion at the knees in spite of the suspension of the child. (*Klose, Jahrb. f. Kinderh.*, 1915, *LXXXII*, 362.)

MUSCULAR HYPERTONIA (Fig. 327).—A condition of general muscular hypertonia has been described by Czerny and Moser.[4] This has been considered as distinct from tetany on the ground that it is persistent; occurs in young infants, usually in the first 6 months of life and especially in the early weeks; is associated with severe disturbance of nutrition, septic states, or syphilis; and is without the characteristic electrical excitability of tetany, although Trousseau's sign may be discovered. Gregor,[5] however, found the electrical reactions present as in tetany. This was not confirmed by Klose,[6] who has made a very extended study of the disease. Some of the cases of prolonged carpopedal spasm may be of this class; but in the more typical cases of hypertonia the contraction generally involves the flexors of the extremities, especially the lower, or may be more widespread and produce retraction of the head and even a degree of opisthotonos.

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[1] Pfaundler und Schlossman, Handb. d. Kinderh., 1906, II, 2, 757.
[2] Amer. Journ. Dis. Child., 1916, XII, 1.
[3] *Loc. cit.*, 403.
[4] Jahrb. f. Kinderh., 1894, XXXVIII, 449.
[5] Monatsschr. f. Psychiat. u. Neurol., 1901, X, 81.
[6] Jahrb. f. Kinderh., 1915, LXXXII, 347.

PSEUDOTETANUS.—In children of from 4 to 10 years of age there is rarely seen a condition designated by Escherich[1] *pseudotetanus*. A case in a child of 1¾ years is described by Hirsch.[2] In this condition there a is rigidity and extreme hardness of the muscles, beginning in some region of the body and rapidly spreading, and including the face and the back, with paroxysmal increase of spasm accompanied by pain. After 3 or 4 weeks recovery gradually follows.

The relationship of this condition as of hypertonia to tetany is not definitely determined, but the existence of intermediate forms indicates their close affiliation or identity with it; although some of the cases probably depend upon other causes. Haas[3] found the frequent association of psychic irritability, vomiting, and other nervous manifestations with the symptoms described, and obtained great benefit from the administration of atropine in full doses.

BRONCHOTETANY.—In this connection reference may be made to the syndrome described by Lederer[4] and entitled *bronchotetany*. He claims it may be the only symptom in spasmophilic subjects. As a result of the spasmophilic contraction of the bronchioles, the alveoli are obstructed and the lungs become atelectatic.

Course and Prognosis.—As an attack of tetany begins to improve the tonic spasm gradually lessens, and the parts may be brought by the patient or by passive movement into the normal position at least for a time, and finally continuously so. The duration is very uncertain. Cases of the intermittent type last in all only a few hours or a day. Then after a short or longer interval another attack may occur. The whole process lasts, as a rule, only a few days or weeks. The persistent form, especially as represented by the carpopedal spasm, may continue for weeks, with little change or with periods of remission. Sometimes the condition yields promptly to treatment, but a decided tendency to relapse is evident. The outcome is never fatal, although death may occur depending upon the primary disease, or upon complications.

Complications.—Omitting those diseases to which tetany is so often secondary, such as rickets and gastrointestinal disturbances, the most frequent complications are other manifestations of the spasmophilic diathesis. Laryngospasm has been described as "tetany of the larynx" and is a frequent attendant; and eclamptic convulsions are very liable to occur in subjects with tetany.

Diagnosis.—This rests upon the bilateral tonic spasm oftenest in the hands and feet; the preservation of consciousness; and, when obtainable, the mechanical excitability and especially the characteristic electrical reactions. *Tetanus* is distinguished by the presence of trismus, and is, moreover, a rare affection even in the new born. Cases of *meningitis* often have the hands in a position sometimes suggesting tetany, but the fingers are usually closed and the mental condition is characteristic. The tonic contractions of some cases of *cerebral diplegia* may be mistaken for tetany, but a careful study of the case will remove all doubt.

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[1] *Loc. cit.*, 416.

[2] Monatsschr. f. Kinderheilk. Referat. 1915, XIV, 394.

[3] Amer. Jour. Dis. Child., 1918, XV, 323.

[4] Zeit. f. Kinderheilk., 1913, VII, 1.

Treatment.—This to be effective and of permanent benefit must be directed to the primary cause. Rickets must receive appropriate treatment, as must especially disorders of the gastrointestinal tract, with the choice of a diet appropriate to the digestive disturbance present. In fact, a proper alteration of the diet is the surest method for the relief of the disease. Feeding with breast-milk is one of the best methods. Cod-liver oil, phosphorus, and general tonic medication are of value. Parathyroid extract and calcium lactate or chloride have each been recommended as a specific treatment of the disease; but their value is still uncertain and the evidence contradictory. There are a number of instances which show a rapid disappearance of the symptoms under the influence of the administration of these. A daily dosage of 2 to 3 grains (0.13 to 0.194) of calcium chloride is recommended (Blühdorn).[1] A number of investigators have found equally beneficial results from the giving of magnesium salts. During the attack itself the patient should be kept at rest, excitement avoided, the bowels opened by a purgative, and efforts made to relieve the spasm by the administration of chloral and the bromides, and by the employment of warm baths.

LARYNGISMUS STRIDULUS

(Laryngospasm. Spasm of the Glottis)

Etiology.—This disease is not a very common one. It is oftenest witnessed in the first 2 years of life, but rarely after this, and not often in the first 6 months. It appears to attack males more frequently; and occurs especially in the early spring and winter months, in this respect resembling tetany. Its relationship to tetany is, in fact, a very close one, and it is believed by Escherich,[2] and others, that most cases of laryngospasm represent only a laryngeal localization of the tonic spasm of this disease. It may be combined with tetany or other evidences of spasmophilia, or be sometimes seen in infants subject to convulsions. Gay[3] in 50 cases of laryngospasm found the facialis symptom present in 47. On the other hand, it may occur without any such characteristic spasmophilic manifestations. The same holds good of its relationship to rickets. It is nearly always only rachitic subjects who suffer from laryngismus stridulus; but exceptions occur and those free from rickets may occasionally exhibit it. Of 50 cases of the disease reported by Gee[4] 48 were unquestionably rachitic. Kirchgässer[5] reported that 89.84 per cent. of 443 collected cases of laryngismus stridulus were also suffering from rickets. Craniotabes is present in some instances, but absent in others; and observed in still others where no spasm of the larnyx occurs; and the relationship of the two is consequently very uncertain. All conditions tending to produce malnutrition with over-excitability of the nervous system may act as predisposing causes of laryngismus. These have been described in considering rachitis and spasmophilia, and need no further mention here. The relation of the disease to the status lymphaticus and enlargement of the thymus gland will be referred to in discussing those subjects (Vol. I, p. 634; Vol. II, p. 521).

As the *exciting cause* of an attack may be any slight disturbance acting reflexly upon the nervous control of the larnyx; such as a sudden fright, anger, hard paroxysms of crying, running about, and overfilling of the stomach; or there may be no cause discoverable and the child may even start from sleep by night or day with an attack.

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[1] Monatsschr. f. Kinderheilk., 1913, Orig., XII, 185.
[2] Traité des mal. de l'enf., Grancher, 1905, IV, 400.
[3] Brain, 1890, XII, 487.
[4] St. Barthol. Hosp. Rep., 1867, III, 103.
[5] Deut. Zeit. f. Nervenheilk., 1900, XVI, 356.

Symptoms.—There are all grades of severity from the mildest to the most severe. There is an absence of fever and of evidences of laryngitis. In the milder attacks the child suddenly stops breathing, the head is thrown back, and the face is pale. This condition lasts only a few seconds, and is followed by a loud crowing inspiration, and the seizure is over. Sometimes the attack begins with several intermittent, noisy inspirations. In the severer cases the closure of the glottis is more complete and long-continued, and all the evidences of suffocation are present. The pallor of the face quickly changes to a deep cyanotic color; the eyeballs project; the expression is anxious; the mouth is open; there are efforts at respiration which are only partially effective or entirely ineffective. Generally the severe cases terminate like the milder in a crowing inspiration, which may be repeated several times before respiration is quite normally established. In still more severe cases there may be unconsciousness with a few clonic spasmodic movements, or even general eclamptic convulsions may develop. Respiratory movements may then return, or the child may die of suffocation. There are other severe cases in which, without convulsions, tonic spasm of the thoracic muscles of respiration suddenly develops, accompanying the larngospasm and producing entire apnea and complete asphyxia without respiratory effort, and the child seems dead and often may, in fact, be so (*expiratory apnea* of Kassowitz).[1] Should recovery from this condition take place, respiration is reëstablished with a crowing inspiration, or quietly if the spasm has passed off completely. The electrical and mechanical excitability of the nerves in laryngismus is similar to that seen in tetany, at least in the cases dependent upon a spasmophilic state.

Course and Prognosis.—The frequency of the attacks varies greatly. They may come at long intervals or be repeated daily or many times a day; in which case the milder attacks often predominate, intermixed with some severer ones. Between or with the paroxysm evidences of spasmophilia may often be discovered. Tetany is present in the majority of cases, and eclampsia is often observed. The duration of the disease may vary from days to months, depending upon the nature of the underlying cause. The prognosis is favorable so far as ultimate recovery is concerned, a gradual lessening in the severity and frequency of the attacks being observed. The danger, however, of death occurring in one of the paroxysms is not to be forgotten. Occasionally this results directly from apnea; in other cases from the development of eclampsia. Yet the majority of cases recover; and it seems probable that many instances of death attributed to laryngismus are dependent upon other causes. (See Lymphatism, Vol. I, p. 632; Diseases of the Thymus, Vol. II, p. 518.)

Diagnosis.—It is to be remembered that the complex of symptoms called "laryngismus stridulus" does not include all instances of spasm of the larnyx. Certain diseases of the brain may be attended by laryngospasm combined with other bulbar or medullary symptoms; or spasm of the larnyx may occur occasionally in the course of meningitis, and is a characteristic symptom of pertussis. A number of other diseases are to be sharply distinguished from laryngismus stridulus, especially to be mentioned being *spasmodic laryngitis*. There is in reality no close resemblance between them. Laryngitis is attended by hoarseness and a characteristic cough; and the attack of stenosis, although so often sudden in onset, is slower in passing. The paroxysm of *pertussis*, as stated, is attended by spasm of the larnyx, and confusion may readily occur in those cases in infancy where the whoop is absent, and the laryngeal closure is the most marked symptom. Always, however, there is some cough, and the course of the disease is in other respects entirely different. The cases of difficult respiration dependent upon *pressure* by an enlarged thymus, mediastinal tumor or abscess, or retroesophageal abscess are slower in onset and in disappearing. Cases of sudden death in the *status lymphaticus* are often improperly attributed to laryngismus. The diagnosis will be considered under that heading (Vol. I, p. 632). Some of the cases of holding the breath (p. 287) are in reality involuntary and are instances of laryngospasm.

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[1] Praktische Kinderheilk., 1910, 266.

Treatment.—Prevention of the attacks is to be accomplished by efforts at modifying the basic cause which is disturbing the nervous system. Particular attention should be paid to the cure of rickets so often present, to the treatment of an underlying spasmophilia, and to the removal of any gastrointestinal disturbance. The diet should be regulated accordingly, breast-milk being provided if possible in cases in the 1st year. An open-air life is of benefit. The administration of cod-liver oil is undoubtedly serviceable, and it may well be combined with phosphorus. Calcium salts are considered a specific by many. (See Tetany, p. 251.) Antispasmodics of various sorts in full doses are indicated, especially the bromides, chloral, and antipyrine. During the attack prompt efforts should be made to relieve the spasm of the larnyx. Taking the infant to the open window, slapping the face with a wet towel or dashing cold water into it, submersion in a warm bath, and similar procedures should be tried to bring about an inspiration. Occasionally, however, methods which cause shock seem to make the condition worse. Inhalation of an anesthetic is useful in severe cases where some degree of respiration is still going on.

CHOREA

(Chorea Minor. St. Vitus's Dance. Sydenham's Chorea)

Name.—According to the interesting history given by Hecker[1] dancing manias arising epidemically under the stress of religious excitement occurred in various places in Europe from the fourteenth to the sixteenth centuries; and in the effort to obtain relief pilgrimages were made to various shrines, among them that of St. Vitus at Zabern and Rotestein, from which fact the title of "St. Vitus's dance" has been derived. There would appear, however, to be no actual relationship between these and the disease now under consideration, which was first clearly described by Sydenham.[2] It is one of the commonest of the diseases of childhood and adolescence.

Pathogenesis.—Lesions have been described in fatal cases of chorea, such as multiple emboli in the capillaries of the brain and other vascular alterations, as well as the changes observed in an accompanying endocarditis. Bacteria of different sorts have been found in the brain in some cases, or in the blood during life, but this has been exceptional. There is no evidence that any of these conditions are in any way characteristic, or necessarily concerned in the production of the symptoms.

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[1] Epidemics of the Middle Ages; Sydenham Soc. Transac., 1844, 87.

[2] Sydenham Society Edition, II, 198; 257.

The exact nature of chorea and its relationships are far from being understood and various theories exist. The disorder is one of the brain and must be regarded as of a functional nature, dependent, perhaps, on temporary interference with the circulation; but no one of the views concerning the methods by which it is brought about appears to apply to all cases. There probably exists an instability of the nerve cells in those suffering from chorea, otherwise the disease would develop more uniformly as a result of the action of some known exciting cause. This instability is supposed by some to be inherited, while the influence of growth has been assigned by others as the cause of it. By still others the disturbance is attributed respectively to psychic influences, cerebral anemia, cerebral hyperemia, irritation of some sort, autointoxication, gastrointestinal disorders, disturbances of nutrition, and infection. The infectious theory has come into especial prominence, and is advocated as a means of explaining particularly the severe cases with evidences of infection elsewhere, as well as the close association of rheumatic endocarditis and chorea. In 2 fatal cases I have recovered bacteria from the cerebral tissues. There is much in support of the theory of infection. Yet so far as rheumatism is concerned, the definite proof that this disease is always an infection by microörganisms is still lacking, and it is, moreover, impossible on this theory to understand the sudden development of chorea after fright and similar mental disturbances. The embolic theory is based upon the existence of the capillary emboli referred to, and is corroborated by the experimental production of chorea in animals by the injection of material in powdered form into the carotid, as done by Money[1] and others. It applies, however, to but few cases, since such emboli are usually not found.

Etiology.—The disease is especially one of the *age* of later childhood and puberty. Of 522 cases analyzed in Philadelphia by Osler[2] 440 (84 per cent.) were between 5 and 15 years of age. Before the age of 5 years there were but 33 cases. It has, however, been seen in infancy, and even congenital chorea is reported, V. Dupuy[3] having collected from medical literature 29 instances in which this diagnosis had been made. The identity of these cases with true chorea is, however, doubtful. The series of Allen Starr[4] is one of the largest published. It includes 1400 cases, of which 73 were in the first 5 years and 1165 (83 per cent.) from 5 to 15 years. In the matter of *sex*, females are more frequently attacked than males, in the proportion of about 2:1 in Starr's series (940 females to 460 males). In the 439 cases of the Collective Investigation Committee of the British Medical Association, as reported by Mackenzie,[5] the ratio of females to males affected was about 3:1. *Heredity* operates only indirectly, as a rule, in that the children of parents of a nervous disposition or with nervous diseases are those most liable to suffer from chorea. Instances of direct inheritance or familial tendency are not very common but have repeatedly been recorded. Osler[6] found that in 80 of 554 cases of his series chorea had occurred in other members of the family, in some instances the parents having had the disease. I have repeatedly seen it in more than one child of a family.

The influence of *season* seems very decided. The larger number of cases occur in the spring months, as pointed out by Lewis[1] in an analysis of 717 separate attacks of chorea from the records of the Philadelphia Orthopedic Hospital and Infirmary for Nervous Diseases during 15 years. The smallest monthly average, 24, occurred in November, and the largest, 114, in March. He is inclined to minimize the influence of school-study, and the anxiety over examinations, which in my own experience has seemed a very positive factor. The series of 226 cases reported by Abt and Levinson[2] does not support the seasonal relationship. *Climate* and *race* are also not without influence. Chorea is distinctly less common in hot climates, and in the Negroes of the United States it is decidedly infrequent. I do not recall seeing more than 2 Negro children with it. It is also rare among the Indians. *Epidemic influence* is not to be overlooked. Although the disease cannot be said to occur in true epidemics, there is no doubt that in certain localities it is much more common in some years than in others. This was true, for instance, in Philadelphia in the winter of 1912. In that year in a ward at the Children's Hospital holding only about 24 beds there were at the same time 11 cases of chorea, and other hospitals of the city showed a similar unusual prevalence. Local epidemics in homes for children may occur, in this case the influence of imitation being apparently decided. Such instances are, however, uncommon.

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[1] Med.-Chir. Transac., 1885, LXVIII, 277.

[2] Chorea and Choreiform Affections, 1894, 6.

[3] Thèse de Paris, 1895.

[4] Jacobi Festschrift, 1900, 5.

[5] Brit. Med. Jour., 1887, I, 425.

[6] *Loc. cit.*,10.

The relationship of chorea to *rheumatism* is very intimate. It is to be remembered in this connection that the manifestations of rheumatism in childhood may be but little marked (see Rheumatism, Vol. I, p. 622), consisting only of slight arthritic pain or stiffness, "growing pains," or, perhaps, of endocarditis or pericarditis without articular manifestations being present or having preceded. Undoubtedly many children who develop chorea have previously suffered from some trivial rheumatic symptoms which have been overlooked. Besides this there are many striking cases often seen in which typical rheumatic arthritis accompanies, precedes, or less often follows chorea in a manner absolutely convincing of the relationship. Thus Batten[3] found that 20 per cent. of those who had had chorea suffered afterwards from rheumatism within 6 years. Concerning the exact proportion of cases of chorea which may be called rheumatic, statistics vary greatly. Sée[4] discovered a history of rheumatism in 61 of 128 cases of chorea; *i.e.*, 47.7 per cent. Osler[5] found that 21 per cent. of his 554 cases gave a history of rheumatism occurring at some time in the family or in the patients themselves. Duckworth[6] considered that 78.5 per cent. of the cases are rheumatic. Of the British Medical Association's series[7] a relationship with rheumatism was reported in 32 per cent. Steiner,[8] however, claims only 1.6 per cent. (4 in 252). If one considers as rheumatism, as I believe should be done, the slight manifestations referred to, the primary cases of endocarditis, and those in which evidences of this disease persist after chorea, the proportion of cases of chorea of rheumatic origin is very large, probably from 50 to 75 per cent.

The direct effect of *psychic causes* is very decided. Often chorea develops promptly after such influences as fright, sudden grief, fear, and the like. It was noted that 285 of the cases of Starr's series appeared to be due to fright or sudden mental shock of some nature. The nervous strain of school life is in my opinion an evident cause. I have seen this especially at the time of year when children are apprehending failure of advancement with their school classes, and this cause seemed to be operative in many instances. Imitation is an occasional psychic factor to which reference has already been made. A number of cases are of hysterical origin. The *acute infectious fevers*, especially scarlatina, are sometimes followed by chorea. There seems no good ground for the claim made that syphilis possesses any etiological relationship. *Reflex influences* of various sorts are assigned as causes but are not a prominent factor. Among those mentioned are diseases of the nose, phimosis, intestinal worms, ocular defects, the approach of the establishment of menstruation, and slight trauma.

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[1] Transac. Assoc. Amer. Phys., 1892, VII, 249.
[2] Jour. Amer. Med. Assoc., 1916, LXVII, 1342.
[3] Lancet, 1898, II, 1195.
[4] Mem. acad. de méd., 1850, XV, 414.
[5] *Loc. cit.*, 14.
[6] Atti d. XI Cong. med. internaz., 1894, III, 354.
[7] *Loc. cit.*, 428.
[8] Ref. Osler, *loc. cit.*, 14.

Symptoms.—All degrees exist from cases of very mild form to more severe and sometimes fatal ones. The onset is usually gradual, the child seeming merely awkward, nervous, restless or emotionally disturbed, and perhaps being reproached by the parents for spilling its food, dropping objects, stumbling when walking, a tendency to cry without reason, making grimaces, and the like. Soon in average cases the jerking movements become so evident that a physician is consulted. Not infrequently, however, they are discovered by the physician on the lookout for symptoms at a time when the parents have not suspected their presence. They begin oftenest on the right side and are most marked in the upper extremities. In about one-third of the cases (449 of Starr's 1400) they are limited to one side of the body (*hemichorea*). Generally, however, even in these some slight jerking may be detected elsewhere as well. Oftenest the movements soon become widespread, involving to some degree all the extremities and the face. They are then very characteristic, taking place without any symmetry or regularity, both when the patient is at rest and during attempted action. They do not occur during sleep, except in very severe attacks. In the cases of but slight severity, the jerking does not interfere much with the patient's acts, and he may sometimes be able to control it for a short time during the examination. I have frequently found that the grasping of the wrist as though the pulse were being felt, or the requesting of the patient to put out the tongue, will reveal the condition. At the wrist there are felt slight twitchings of the muscles, while the tongue is often suddenly jerked back into the oral cavity before the order is given to close the mouth. Usually, however, there is no need for such a procedure, since the movements are very apparent, being made worse by excitement or fatigue, or by efforts to control them. Some degree of psychic disturbance is present. Sleep is generally poor and restless, with dreaming.

In many instances the jerking and the making of grimaces are nearly constant and the patient is unable to feed himself or dress, or even to walk. The respiration is jerking and irregular from the involvement of the diaphragm. Speech, too, is affected and the patient either talks in a hurried, interrupted, stumbling manner or may not be able to articulate at all. Some involvement of speech was observed in 556 of Starr's 1400 cases. Some of this interference with speech in the severe cases appears to depend upon the disturbed psychic state, and the inability to think connectedly. There is often present a loss of power in some of the limbs, the condition simulating paralysis. This is especially deceptive if the paresis affects chiefly one limb or one side of the body.

Sometimes the paresis is much in excess of the jerking movements (*paralytic chorea; chorea mollis*).

The great majority of cases do not pass beyond the condition described. Occasionally, however, a choreic state of frightful intensity is observed (*chorea insaniens*). In this the movements are so severe and persistent that the child is tossed violently about the bed and becomes covered with bruises and abrasions from striking against the frame work of the bed-stead, and forcible restraint is required. They continue during the night, and there is almost no sleep. Speech is almost or quite impossible, and swallowing is difficult. The expression of the face is that of a maniacal person.

The tactile sensibility in chorea is usually not disturbed. Headache may occur; pain in the limbs is very frequent; paresthesia is exceptional. The reflexes are variable, sometimes exaggerated and sometimes diminished. The nutrition is poor; a loss of adipose tissue occurs; there is a general diminution of muscular tone especially in the limbs most affected; the patients are easily fatigued, and anemia develops. With regard to the latter, however, it is possible that in many instances the anemia is apparent only, and a blood-count should be made to determine this (Burr).[1] The appetite is much diminished in the severer cases, and in the worst the sphincters may be involved. The action of the heart is often accelerated, and murmurs may be present; often soft, blowing and dependent upon anemia or, according to some views, on chorea of the muscles of the heart, and therefore functional and temporary; still oftener the result of a developing endocarditis and persisting after recovery from the chorea.

Fever of moderate degree is sometimes observed, but this usually depends upon a complicating endocarditis or some other condition. Except under these circumstances, or in the severest cases, it can hardly be called a characteristic of the disease. The urine commonly exhibits an increased amount of urea; albuminuria may occur in the very severe cases; and glycosuria has been recorded. Uro-hematoporphyrin was found by Garrod[2] in 14 out of 20 cases. The cerebrospinal fluid has exhibited a lymphocytosis in some instances. Richardière, Lemaire and Sourdel[3] found this in all of 15 cases examined. The occurrence of psychic disturbances has already been referred to. They are nearly always present to some degree and constitute an important element of the disease. They were noted in 827 of Starr's series of 1400 cases. Underlying all is a condition of psychic irritability and lack of balance which may show itself only in fretfulness; willfulness; capriciousness; sudden attacks of crying or laughing without cause; lack of memory and mental concentration. In the severer attacks this may increase to a certain degree of stupidity, as indicated by the expression of the face which is that suggestive of imbecility; or may advance still further to violent maniacal excitement in the cases of the worst grade. In addition, a number of other psychic disturbances may exceptionally be observed, such as melancholia and hallucinations.

Course and Prognosis.—The disorder requires 1 or 2 weeks to reach its height; then remains stationary for a time; and gradually grows less and disappears after from 8 to 10 weeks or sometimes several months. Exceptionally the chorea takes on a chronic form and may last for years, generally with but few symptoms, and then finally recover. The prognosis, as regards the chorea itself, is good in the great majority of cases. The psychic disturbances disappear with the choreic movements, and all traces of paresis are presently lost. The severest cases may die from a complicating endocarditis or cerebral lesion, or worn out by the violence of the movements. The death-rate for all cases may be placed at 2 to 3 per cent. Most of the fatal cases occur after puberty.

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[1] Univ. Med. Magaz., 1896, IX, 188.

[2] Lancet, 1892, I, 793.

[3] Annal. de méd. et de chirurg. inf., 1911, XV, 276.

Always the prognosis for perfect recovery must be guarded, on account of the danger of the development of endocarditis or pericarditis with permanent cardiac lesions resulting. The frequency of cardiac complication cannot be easily determined, at least as based upon the development of systolic murmurs during the attack; owing to the impossibility of deciding in cases which recover without a cardiac murmur whether that heard during the course of the disease was functional, or was due to a transitory endocarditis. Osler[1] found out of 140 cases of chorea examined more than 2 years after the attack the evidence of persisting chronic disease of the heart in 72; *i.e.*, 51.44 per cent.; and in 73 collected autopsies on fatal cases found 62 with recent lesions of endocarditis. In the 439 cases of chorea of the British Medical Association's series[2] 32 per cent. exhibited a decided cardiac lesion, and in 16 per cent. there was present during the attack some cardiac derangement believed to be functional; and Abt and Levinson[3] in 226 cases of chorea found a cardiac involvement in 32 per cent. The frequency with which rheumatism is associated with chorea either as an antecedent or a sequel has already been alluded to. Tonsillitis is a common complication, and nephritis is occasionally observed.

Relapse and Recurrence.—The possibility of relapse and recurrence in chorea is very decided. The former is seen less often; but the tendency to recurrence is well known. Of 410 cases in Osler's statistics[4] only 240 had but 1 attack and 60 had more than 2. These recurrences are liable to come on in the springtime at the period when the greatest disposition to the disease exists.

Diagnosis.—There is scarcely any condition which can be confounded with typically developed chorea. The jerking, arrhythmical movements are entirely characteristic. Nevertheless in less well-marked instances mistakes in diagnosis can and often do occur. The movements of *post-paralytic chorea*, sometimes seen in cases of cerebral paralysis, are generally unilateral and chronic in their course. The intelligence is often not normal. The affected limbs show a condition of spastic rigidity, with more or less loss of power. In some instances the movements are bilateral and very marked, depending upon a lesion of the cortex upon both sides with subsequent sclerosis. The resemblance to chorea in such instance is at first quite close, but the history of the case and the other symptoms present serve readily to distinguish the disease. The paralysis of *poliomyelitis* bears a likeness to paralytic chorea in its flaccidity; but differs in the history of the disease, the atrophy, and the reaction of degeneration. I have seen *Friedreich's ataxia* diagnosticated chorea, but the resemblance is, in fact, only slight. The ataxic movements of this disease are not so quick and jerking; there is a characteristic slowness of speech; nystagmus; and a peculiar habitus.

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[1] *Loc. cit.*, 55; 60.

[2] *Loc. cit.*, 431.

[3] *Loc. cit.*, 1342.

[4] *Loc. cit.*, 40.

(See Friedreich's Ataxia, p. 383.) *Hysteria* is not often a cause of difficulty in diagnosis in early life. The movements are usually more rhythmic in character, and although they may sometimes be arrhythmic and closely simulate those of chorea, the history of the case and the presence of distinct evidences of hysteria serve to distinguish. *Huntingdon's chorea* is not a disease of childhood, and is a chronic familial disorder progressive to a fatal termination. *Habit spasm* or habit chorea belongs properly to the class of tics. Some of the cases simulate the minor instances of chorea at first very closely, and only repeated examination and an accurate history will serve to distinguish. The course, however, is essentially chronic and the movements are limited to certain regions and can be restrained by the efforts of the child, to begin again when the control is removed. Finally is to be mentioned *chorea electrica* which resembles chorea minor in name only. The movements are peculiarly sudden and the speech is unaffected.

Treatment.—To **prevent** chorea in those of a neuropathic tendency, excitable disposition, or who have had previous attacks of the disease, very careful regulation should be made of the general life. All over-study at school should be carefully guarded against. Undue excitement is to be shunned. The diet should be light and digestible to avoid the possible influence of autointoxication. The general health should be maintained by tonics or other remedies which may be indicated. So, too, those who have previously had rheumatism should have all possible measures employed to prevent exposure to cold and damp in order to avoid recurrence, lest chorea also develop.

For the **attack** itself the most important treatment is hygienic. School should be stopped at once. Even in the mildest cases association with other children is to be avoided lest ridicule and the over-excitement of play make the choreic condition worse. No thought of punishment or of scolding should be entertained by the parents. Faulty digestive processes should be corrected and the diet made plain and assimilable, with the omission of tea, coffee or alcoholic beverages. Iron is to be administered if anemia is present. A life out of doors is most desirable, but this must be obtained without much exercise. In fact, one of the most successful methods of treatment is rest in bed. This should be insisted upon in all but the mildest cases, and even in these it is advisable. Rest of body should be attended by rest of mind as well, and visits by members of the family or others should be forbidden or curtailed. In hospital practice patients with chorea may often be screened or kept in separate rooms with advantage. With regard to isolation, however, judgment must be used, since it is possible for too strict a seclusion to produce unhappiness and fretting, and thus do harm instead of good. How long the confinement to bed shall continue is an open question. My own custom is to allow the child to be up when the movements have become decidedly better, without having entirely disappeared, although the exercise taken should still be much curtailed. In suitable weather the bed should be kept out of doors, if a satisfactory place offers. Daily warm tub-baths or warm packs are often very beneficial, and gentle massage is also sometimes of service. The worst cases of chorea demand, necessarily, forcible restraint, or the padding of the bed-stead to avoid injury of the body; and the employment of a water-bed is often necessary to prevent bed-sores. Throughout the whole course of an attack of chorea the heart should be very carefully watched, and even the mildest cases put to bed on the first development of a murmur; while the duration of confinement of cases already there and the absence of excitement is to be lengthened.

The employment of drugs intended to control the disease is not very satisfactory. Arsenic has long enjoyed a repute. It may be commenced in doses of 3 minims (0.18) of Fowler's solution 3 times a day, well diluted, after meals, and gradually increased in the course of a week to 10 minims (0.62) 3 times daily, if no evidence develops of any disturbance of digestion, puffiness about the eyes, or albuminuria. The arsenic in this maximum dosage should be continued for a week more, and then the administration stopped for 3 or 4 days, after which the full dosage may be recommenced and continued for another 2 weeks. Arsenic has also been given in the form of arsphenamine by Talent,[1] Bokay[2] and others, and rapid improvement or decided curtailing of the course of the disease has been reported. Yet the administration of arsenic is not without danger. I have seen well-marked arsenical neuritis develop, and other such cases have been reported; or pigmentation of the skin may result. As to the real value of arsenic or any other drug in curtailing the duration of chorea there is room for doubt, and opinions are not uniform. The disease has a natural tendency to cease of itself, and the recovery attributed to drugs may be, in fact, in no way due to them. Among other remedies recommended, antipyrine often lessens the severity of the movements without evidence that it is curative of the disease. A dose of 1 grain (0.065) or even more per year of age, given 3 times a day or oftener, may be administered safely. In other cases the bromides serve the same purpose. The salicylates have been highly praised for cases associated closely with rheumatic manifestations, and for the purpose also of preventing the development of endocarditis; and in such instances they should certainly be tried, although the benefit to be expected is problematical. In the severest cases the wet pack may be advantageously employed and such remedies as chloral, morphine, veronal, and hyoscine hydrobromate may serve to lessen the violence of the movements and produce sleep. I have had encouraging results with the last-mentioned drug. Among other remedies recommended are apomorphine, magnesium sulphate intraspinously, and the employment of simple lumbar puncture. It is important, too, in severe cases not to delay the beginning of supporting and stimulating treatment. A method of treatment proposed by Goodman[3] consists in drawing blood from a vein of the patient, separating the serum, and injecting this into the spinal canal after lumbar puncture, and after the withdrawal of an equal or larger amount of the spinal fluid. Very brilliant results are reported. My own results in the few cases tried have not been very satisfactory. Porter[4] used intrathecal injection of horse-serum in severe cases, with what appeared to be undoubted benefit; but is not sure that the psychic influence of the procedure was not the chief agent.

After recovery from chorea a period of several weeks should elapse before the child is permitted to resume school life or other exciting conditions; and care must be taken also to improve the strength and remove any anemia which may remain. For these cases, and for those which are proving refractory to treatment, a change of air may be of much benefit.

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[1] Thèse de Paris, 1913.
[2] Deut. med. Woch., 1911, XXXVII, 111.
[3] Arch. of Pediat., 1916, XXXIII, 649.
[4] Amer. Jour. Dis. Child., 1918, XVI, 109

SPASMUS NUTANS

(Nodding Spasm; Gyrospasm)

Etiology.—This, a not common condition, was first described by Eberth.[1] Raudnitz[2] recorded but 14 cases in 52,213 sick children in the Buda-Pest Children's Hospital. It occurs chiefly in infancy, and especially from the age of 4 to 12 months, although cases beginning in the 2d year or even later are occasionally seen. It is somewhat more common in females. The causes are various and their method of action not clearly understood. One of the most important predisposing factors is rickets, and almost all the cases observed in infants are associated with this disease. The majority of the instances are seen in the winter. In 95 cases analyzed by Thomson[3] 68 began in the months of December, January and February. This is probably due to the lesser amount of light in these months; since residence in dark houses and streets appears to be a very important cause of the disease, the child having to turn its eyes obliquely or upward in order to see distinctly such objects as toys and the like. Any debilitating disorder may aid in producing the symptoms, as may likewise falls upon the head. The association of the occupying of dark rooms with the development of head-nodding was first pointed out by Raudnitz.

Symptoms and Course.—The symptoms consist in either an involuntary nodding of the head, or a rotary movement of it from side to side. The range of movement is slight, and the rapidity about once a second or slightly faster. The movements may be constantly present or only discoverable at times. They increase with the efforts to fix the eyes upon an object, and disappear during sleep or if the eyes are bandaged. Sometimes they are observed only when the light comes in a particular direction. The infant often has the habit of holding the head obliquely or bent a little backward.

With the shaking movements of the head there is nystagmus, either of one or both eyes. This may first develop at the same time with the movements of the head, or before or after them. The movements of the eyes are rapid, of slight extent, and may occur in any direction, varying often at times in the same case. Forcible fixing of the head increases the nystagmus. In some cases there is an attendant convergent strabismus. The intelligence is normal and the disease has no effect upon the general health; which, however, is usually previously debilitated. The duration is several months as a rule. Occasionally recurrence is observed, or the condition may drag on for a year. The prognosis is entirely good.

Diagnosis.—There is no difficulty attending this. The age of the patient and the absence of all other symptoms than those described serve to distinguish the disorder. *Congenital nystagmus* is accompanied by ocular defect, and is without the movements of the head. *Eclampsia nutans*, to which, and not to nodding spasm, the title "Salaam convulsion" is properly applied, is a rare affection of older subjects, usually without nystagmus, and attended by psychic or hysterical manifestations. The *head nodding* which occurs more as a habit (p. 286) is seen in older children, is not attended by nystagmus, and has a much larger range of movement. The *rocking* of the head on the pillow observed so often in rachitic infants is an entirely different affection, slower in movement, and attended by symptoms of fretfulness and irritability.

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[1] Charité Annalen, 1850, I, 753.

[2] Jahrb. f. Kinderheilk., 1897, XLV, 145.

[3] Traité des mal. de l'enfance, Grancher, 1905, IV, 278.

Treatment.—Therapeutic measures consist in remedying the insufficiency of light and other bad hygienic conditions in the dwelling, and in treating the rachitic or other debilitating disorders present. Antispasmodics such as antipyrine or the bromides may be administered, but are scarcely required.

THE TICS

These include a number of affections, bearing some resemblance to chorea. Certain of them are probably allied to hysteria.

1. HABIT-SPASM

(Habit Tic; Simple Tic; Habit Chorea)

Etiology.—This, the most frequent of the tics, is a common condition seen oftenest in early and later childhood. Of 143 cases studied by Sinkler[1] 109 first developed symptoms between 5 and 15 years of age; 74 were males and 69 females. It occurs especially in those with a neuropathic disposition or inheritance, and develops oftenest when the general health is below normal, or when some such cause as overwork at school or similar mental strain is operative. There is frequently also a local cause, such as adenoids, defective vision, or localized pain or sense of discomfort in some region, which determines the position and character of the movements. Occasionally imitation is the starting point of the disease, while in other instances the movements of habit-spasm may persist after recovery from a chorea.

Symptoms.—These consist in movements oftenest connected with the face; such as twitching or distortion of the mouth, wrinkling of the forehead, elevating the eyebrows, forcible winking, and the like; or there may be sighing or sniffing, or jerking movements of the whole head. The movements are of a spasmodic nature; occur at irregular intervals; are only occasional at first but soon become very frequent; are made worse by excitement; and are done unconsciously, although the child is able to restrain them by force of will. Sometimes the effort at control produces such a sensation of discomfort that the patient is unwilling to make it. Other cases have jerking movements of the hands or the body, especially of the arms and shoulders, while others exhibit repeated forced efforts at coughing. There may be only one sort of movement, or a number of various kinds; or one sort may be replaced by another.

Prognosis and Diagnosis.—The duration of the disease is very uncertain. Sometimes it lasts but a few months, often to recur if the general health again suffers or some other cause becomes active. In other cases it may be very persistent, lasting for years or perhaps for a life-time. The diagnosis is usually easy. Generally the much longer duration and the limitation of the movement to the face or to some other part of the body serve to distinguish it from chorea. When the arms as well as the face are involved, the diagnosis from chorea is not always easy at the first examination.

Treatment.—This is not always satisfactory, especially in cases which have persisted for some time. It is very important to enlist the efforts of the child to guard against and control the habit. The parents should call the attention of the patient to the matter often enough to encourage his efforts, but not so frequently that this degenerates to nagging or makes the child over-conscious. Very careful discrimination must be used in this matter or damage instead of good results. A careful search is required for all possible exciting causes, especially the presence of adenoids or other nasal disease, and the existence of defective vision, and the proper treatment instituted. Particular attention must be given to the general health. In some cases the insisting upon rest recumbent a portion of every day is of service. As far as drugs are concerned, arsenic appears to be the most beneficial. Strychnine is also recommended.

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[1] Amer. Jour. Med. Sci., 1897, CXIII, 559.

2. IMPULSIVE TIC

(Gilles de la Tourette's Disease)

This condition, studied especially by Gilles de la Tourette,[1] and often bearing his name, possesses some resemblance to habit spasm in symptoms, but is more closely allied in nature to hysteria and to pyschasthenia. It begins oftenest in later childhood or at puberty, is of very much less frequency than the malady just described, and generally exhibits a marked neuropathic inheritance.

The **symptoms** consist in convulsive twitchings, especially of the muscles of the face and arms, but in some cases of other parts of the body as well; and these may be very violent. With the movements is associated the production of explosive sounds, such as a loud barking cough; or the enunciation of certain words. A frequent symptom is the tendency to repeat several times a word just heard (*echolalia*) or the involuntary uttering of foul language (*coprolalia*). This is generally done at the same time as the impulsive movements take place. In many cases imperative conceptions are associated with the movements, resulting in the child performing certain acts, such as the touching of some object; sudden jumping; counting a certain number of times; the making of hissing sounds; the speaking of a number of words in sequence, and the like.

The **course** is very chronic, and the prognosis for recovery is not favorable, although the disease occasionally disappears. The **diagnosis** is to be made from chorea and from habit spasm. The movements are not so frequent as in chorea and are not so irregular, and this latter disease is without the explosive utterances and the imperative conceptions. Habit spasm is like impulsive tic in many of the movements, but does not reach the same degree of violence, and is without the other symptoms of the latter disease.

The **treatment** is generally unsatisfactory. It consists in efforts to improve the underlying neuropathic disposition. Isolation and change of scene, hydrotherapy, gymnastic exercises, and measures to better the general health should be instituted. Suggestion may be of value in cases possibly of a hysterical nature, together with other remedies suitable in that disease. The patient should be taught self-control; but not too much notice should seem to be taken of the spasmodic attacks.

3. CHOREA ELECTRICA

Several forms of this exist, all of them of rare occurrence, and none of them seen before later childhood or puberty. The disease appears to belong to the tics, but in some instances may be a manifestation of hysteria, or possibly of epilepsy. One form described by Dubini,[2] seen in adults as well, exhibits a series of violent, frequently repeated, convulsive movements of various parts of the body, especially the head and neck, resembling those produced by a strong electric current. Epileptiform convulsions also occur and paralysis finally develops, with wasting of the muscles and loss of faradic contractility. There is much pain, but generally no persistent loss of consciousness until the end, when coma may appear. The disease ends fatally in a few weeks, or less often not until after a year. The nature is unknown, but the malady has been supposed to be of infectious origin. It is confined chiefly to Italy.

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[1] Arch. de Neurol., 1885, IX, 19.

[2] Giorno di Milano e Gaz. med., 1849. Ref., Bruns., Berl. klin. Wochenschr., 1902, XXXIX, 1185.

A second form was described by Henoch[1] in children of from 9 to 15 years of age. In this variety there are sudden, shock-like contractions of the muscles of the head and neck, or less often of other parts of the body. Each movement is of a momentary duration and generally slight in degree. They may be repeated at intervals of seconds or minutes, and occur by day and occasionally also during the night. Speech is unaffected, and the control of the muscles of the body undisturbed except at the moment of the jerking. The cause is entirely unknown, the duration is of long continuance, and the disease uninfluenced by treatment.

A third form was observed first in children under the care of Bergeron, and was described by Berland in 1880.[2] The movements are similar to those of the variety just described. The patients are generally anemic, debilitated, or nervous, or at least of a nervous ancestry. The onset is usually sudden, sometimes following a nervous shock of some sort; and, unlike Henoch's type, the prognosis is good. All this makes the disease closely allied to hysteria, and perhaps a manifestation of it.

The relationship of these varieties of electric chorea to each other and their true nature are uncertain. The **diagnosis** is to be made from habit spasm by the sudden shock-like character of the movements; from impulsive tic by the absence of echolalia and, in the last two forms, the lesser excursus of the movements and the absence of mental disturbance. Chorea minor exhibits more widespread jerking of a more irregular and less sudden character, and speech is often characteristically involved. Paramyoclonus multiplex has perhaps the closest similarity of appearance to electric chorea; but movements involve especially the extremities and are symmetrical, and there are sensory disturbances. Yet paramyoclonus and the electric chorea of Henoch are held by many to be identical.

NERVOUS CHILDREN

(Neuropathic Children; Nervousness; Neuropathic Diathesis)

In a broader sense this term includes children suffering from one or more of a number of different conditions such as disturbances of sleep, headache, neurasthenia, bad habits, and hysteria. It does not follow, however, that all children with some of these manifestations are necessarily to be classed among the simply "nervous;" and for this reason these various individual topics referred to will receive independent consideration.

Etiology.—In the narrower sense here employed, there are many children who, without necessarily suffering from any distinct nervous disease of moment, are to be denominated *nervous*. (Simple endogenous nervousness of Cramer).[1] Usually there is an *inherited tendency* to nervousness, one or both of the parents or other members of the family being distinctly of this character. There is this underlying neuropathic basis present in most cases, sufficient alone to produce the symptoms, or to permit of their developing or growing worse under the influence of *exciting causes.* Among these causes are the natural physiological and anatomical peculiarities of early life. There is, in the first place, the incomplete development of the centers in the cerebral cortex, with consequent lack of control over reflex excitations. There is, further, the rapid growth of the intellect and the interest in the outside world, without a balancing experience and judgment. These conditions favor the development of nervous symptoms in those with or without inherited nervous disposition. There is also the very strong tendency to imitation seen in early life, which may readily give rise to pathological nervous states. Further, with the imperfect establishment of the final relationships which will exist later, there is combined an amount of mental and physical strain undergone by children from early infancy through the school years which is enormous, and the evil influence of this on many subjects is great; since a child will exert itself physically at play until absolutely tired out, or will be allowed to spend many more hours of confinement at mental work in school than many of its seniors could well endure. This overstrain of body and of mind in the absence of proper control by the parents, or even abetted by them, is a very important cause of the development of nervousness. The constant association with nervous excitable relations is another cause, as are the efforts to make an infant learn quickly, or to show, in general, evidences of precocity. Many infants are kept up late at night or disturbed at other times for the benefit of the older members of the family or of visitors; with forgetfulness of their absolute need of many hours of sleep and of much mental quiet at other times. On the other hand, one often sees the development of nervousness in an only child who is pampered and overguarded by the parents, mingles only with older persons, and is without that association with other children which is so important for proper mental development.

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[1] Vorlesungen ü. Kinderkr., 1895, 193

[2] Ref. Bézy in Grancher, etc., Traité des mal. de l'enf., 1905, IV, 377.

The influence of school life has already been referred to. Undue mental labor; worry about unfinished lessons and imperfect recitations; the strain of keeping near the head of the class; the fear that he will fail to pass examinations; are all powerful influences for the development of nervousness in a child predisposed to it. Many of the teachers, and still more the parents, are to blame. There should be no method which forces children to keep the same pace in the same studies. Lastly may be mentioned shock, fright, trauma of any sort, or the influence of some debilitating disease, which often is the direct cause of the development of a condition of nervousness.

Symptoms.—The symptoms seen in the nervous child are very varied and differ with the individual. They may appear even in the 1st year of life. At this time are observed, for instance, such manifestations as vomiting produced by such causes as slight emotional excitement or a trifling alteration in the taste of the food; the refusal to take any new article of diet (see Anorexia Nervosa, Vol. I, p. 706); starting from sleep from very insignificant causes, such as a slight noise or light in the room; precocious development in various directions; the early acquiring of unusual fear of or great shyness at the approach of strangers; thigh-friction; very active emotional facial expression; excitement without sufficient reason; and the like. Babies of this class are often subjects of imperfect nutrition shown in various ways.

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[1] Bruns, Cramer, u. Ziehen, Handb. d. Nervenkrankh. im Kindersalter, 1912, 22.

Older children of the neurotic type frequently are delicately built, anemic, easily tired mentally and physically, suffer readily from digestive disturbances, and have capricious appetites. There may be a tendency to cardiac irregularity or rapid respiration. Nervous coughing or vomiting is sometimes seen following any excitement. Some of the stigmata of degeneration to be described later (p. 296) may be present, but are by no means necessary accompaniments; or, on the other hand, may exist without any evidence of nervousness. These nervous children are very impressionable, emotional, and easily excited, and express this to a degree in excess of that seen in normal children. In some cases there is an unusual movement of the face during speech, with the production of grimaces. The power of imagination, always great in a child, is unusually developed, and may be the cause of terror at night from the images which the mind creates, and often of fear of remaining alone or without a light. Nervous children are disposed to worry or fret; are timid; unduly anxious over minor matters; and in some instances have an uncontrollable fear of harmless animals and other objects. Sometimes this very characteristic tendency to fear or dread is originally awakened by ill-considered remarks of, or a story told by, the parents, nurses, or companions. In other instances it seems to arise chiefly from the child's power of imagination. Nervous children are sensitive, and slight reproach or reproof is the cause of unhappiness and morbidity. This self-consciousness makes them shy with strangers and even with associates. Various idiosyncrasies are liable to develop as a result of the nervous condition. At school and at home the children may be conscious of the difference between themselves and others, and they may become self-repressed, reticent and silent; self-conscious and brooding. Headache is a common and troublesome symptom.

The intellectual power of the nervous child is often unusally great, and the mastering of the school work is a matter of no difficulty. In other cases the power is only normal, and the vanity of the parent or child makes preëminence at studies attained only by undue effort. In very many the intellectual power becomes easily fatigued on slight mental exertion, and the child finds it difficult to keep up with classmates at school.

Course of Prognosis.—The state of nervousness may be of but temporary duration when the exciting cause was a rapidly acting one, such as trauma or acute illness. When once thoroughly established, or of long existence, only prolonged treatment can be of avail. If not properly cared for, the condition readily passes beyond the domain of mere nervousness into that of neurasthenia, psychasthenia, psychoses, or other nervous disorder. Yet the prognosis for nervous children is not entirely unfavorable under proper management. The underlying neuropathic tendency, present in practically all cases and usually inherited, always remains, but may cease to give trouble if the life is properly regulated. The outcome often depends on the care and exactness with which the treatment can be carried out; and especially on how well the parents can be instructed in the proper management of the child. It is to be remembered that the strain thrown upon these children must continue always to be in accord with the diminished resisting power which is characteristic of them; otherwise at any time during life there may be a return of nervous symptoms.

Diagnosis.—There is much confusion in the conception and definition of some of the forms of neuropathic and allied disorders, and a sharp differentiation cannot always be made. This applies especially to simple nervousness, neurasthenia, hysteria, and psychasthenia. They appear to shade into each other, or to be variously combined, often making the diagnosis difficult. The diagnosis of simple *nervousness* rests upon the congenital origin usually present; the absence of intellectual defect; the easily developed mental and bodily fatigue; the tendency to excitability and timidity; and the other symptoms as detailed. *Neurasthenia*, as it occurs in children, does not necessarily possess any congenital origin but may be acquired. In the stricter sense, as employed by Cramer[1] it is characterized by the excessive degree of weakness and irritability of mind and body. *Psychopathic children* may exhibit some of the characters of the neuropathic, but with the addition of a great predominance of psychic manifestations. The condition is congenital in origin. Some of the stigmata of degeneration are liable to be exhibited. The state of the intelligence is variable. *Hysteria* is characterized by defective will-power, emotional excitability, and the control of the body and mind by perverted notions and fixed ideas, which are not uncommonly produced by suggestion (Rachford).[2]

Treatment.—This is difficult and tedious. From early infancy the evidence of a neurotic disposition must be watched for and the development or increase of symptoms guarded against by proper education and control. Late hours, broken sleep, excitement of the young baby by visitors, and other disturbing causes are to be carefully shunned. Later in infancy great caution must be used against forcing the baby into precocity by strenuous efforts to teach it to talk or walk. Both at this period and after it there must be an abundance of mental and bodily rest and of sleep. It is especially in the school years that constant supervision is required, and most careful judgment used in the management of the nervous child. A middle ground must be chosen between too much and too little study. The child must necessarily receive instruction, and, too constantly keeping at home from school but makes him lazy and indifferent. This must never be permitted. It is very important to obtain association with other children, and the removal of a child entirely from school gives him no occupation and no playmates, and makes him feel that he is an invalid. The question of schooling is thus a most difficult one, and is purely an individual matter. Most important is it that the parents shall cease to pamper a nervous child, or to yield to all his whims, supplying specially prepared articles of diet according to his fancies, and constantly showing a very manifest anxiety over his condition. This concentrates his thoughts upon himself. It is a common habit of parents to show their anxiety and to detail a child's nervous symptoms to the physician in the presence of the patient. This must never be allowed.

One of the most vital elements of treatment is, unfortunately, one which frequently cannot be fulfilled; viz., the removal entirely from the nervous influence of home associations. The sending of the child to a boarding school is often greatly to be desired. In other instances, the spending of the summer vacation where there are new sights and associations is of very great benefit; localities always being selected where there are other children to be had as playmates. The direct influence of change of climate is in itself beneficial.

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[1] *Loc. cit.*, 21.

[2] Neurotic Disorders of Children, 1905, 328.

Naturally, every method possible must be employed to improve the general health; such as properly chosen gymnastic exercises; life in the open air; exercise out-of-doors; suitable diet; and the treatment of any disordered condition present. After the occurrence of any acute illness, there should be no haste in returning the child to school; since the strength of nervous children often suffers decidedly during the attack. Throughout there must be encouragment given, and the symptoms openly made light of rather than dwelt upon. The unreasoning timidity which is usually present is to be managed carefully. Thus a nervous child who has developed, for instance, a fear of sleeping in the dark, should be allowed a light in the room, and that without question. Nothing can be accomplished by compulsion in such cases; but encouragment will aid greatly. Disciplining nervous children is, of course, required on occasions, or they become self-willed and uncontrollable; but such forms must be chosen as cannot awaken fright or other nervous shock, lest the nervous symptoms are made worse thereby.

NEURASTHENIA

In the condition of Nervousness, as just described, are included many of the cases designated by some writers as neurasthenia. In the narrower sense here employed, following to some extent the definition of Cramer,[1] neurasthenia in children is an uncommon affection.

The **cause** may be an underlying congenital predisposition, but this does not appear to be necessary, and the disorder may come on in children previously healthy, produced by excessive over-work at school or in other ways, and being then acquired. The condition is a chronic one and the symptoms are to some extent those seen in nervous children; but especially marked is a profound exhaustion of the mental and bodily powers, very often combined with severe prolonged headache and attacks of fainting. The children are pale, anemic, and often with the nutrition much impaired, and complain of great fatigue. There is frequently loss of appetite, constipation or diarrhea, insomnia, depression, irritability, or easily aroused emotions. In some cases there is increase of the tendon-reflexes with diminution of those of the throat and cornea; trembling of the eyelids and hands; inequality of the pupils, and stammering speech. The intellect is unaffected except for the mental asthenia which renders long-continuance of thought impossible.

The **duration** of the disease is long, even under favorable circumstances, and some years may be required in severe cases before recovery is obtained. The affection is, however, a curable one.

Treatment consists primarily in removing the causes of the exhausted nervous condition. Great physical and mental repose is required. Rest in bed may be needed for a time, with removal from all exciting surroundings. Then change of air and scene are advisable, with massage and hydrotherapeutic measures. Constant encouragement of the patient is necessary in order to remove the mental depression.

HEADACHE

Etiology and Symptoms.—Headache, although but a symptom, may be dependent upon so many and such diverse conditions that some review of its causes and indications is necessary. It is probably not common in infancy, although this is something which cannot be determined with absolute certainty; and in early childhood it occurs less frequently than later. Among the different varieties, some of them distinctly overlapping, may be mentioned the following:

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[1] Bruns, Cramer, and Ziehen, Handb. d. Nervenkrank. im Kindersalter, 1912, 21.

1. **Headache Attendant upon Organic Diseases of the Brain or Meninges.** This is seen in various conditions, such as intracranial tumor, meningitis of various forms, poliomyelitis, abscess of the brain, sinus-thrombosis, and syphilis. The pain is more or less persistent, frequently severe, and localized. In meningitis it is often situated in the occiput.

2. **Headache with Infectious Diseases.**—Headache is common among the prodromal symptoms of many infectious diseases, especially typhoid fever, scarlet fever, and diphtheria. The frontal situation, the lack of persistence for more than a few hours or days, and the association of fever and other symptoms are aids to the correct diagnosis.

3. **Toxemic Headache.**—Especially to be mentioned here, as of very common occurrence, is that dependent upon gastrointestinal disorders. This is frequently denominated "bilious headache," being associated with distinct symptoms of indigestion, as vomiting, coated tongue, loss of appetite, malaise, and often constipation. This form of headache is generally frontal or vertical. Much less frequent in children is the headache resulting from the toxic influence of metallic or other poisons, such as lead, alcohol, opium, carbonic dioxide and the like.

4. **Headache Due to Disturbance of the Cerebral Circulation.**—Most prominent here, and very often observed, is that dependent upon anemia. This headache is frontal or oftener vertical, dull, and most marked in the morning. The presence of distinct signs of anemia and other symptoms readily produced by this will be noticed. Hyperemia of the brain is likewise a cause of headache (*congestive headache*). This may sometimes be seen in pertussis, cardiac disease, difficult or delayed menstruation in girls approaching puberty, sunstroke, and intense mental activity. It is sometimes attended by flushing of the face and injection of the eyes.

5. **Nervous Headache.**—This is a marked symptom in the case of the nervous children already described (p. 269). It may be brought on or increased by mental work or bodily fatigue. Among other causes are hysteria, neurasthenia, and epilepsy. This headache is liable to occur day after day, and is frequently vertical. Many of the "school headaches" are of this nature. Anemia and insomnia are often attendant symptoms.

6. **Headache Depending upon Disorders of the Special Senses.**—The most frequent variety of this class is the headache due to eye-strain. It is frontal or occipital in position, and is prone to develop after the eyes have been used for study, or after prolonged exposure to bright light. Conjunctivitis or keratitis is present in some cases. Disorders of the nose, such as adenoids, rhinitis and polypi, may be a reflex cause of headache; and otitis may produce an intense pain in the parietal or temporal region.

7. **Neuralgic Headache.**—This is produced by many of the causes mentioned, but is not as common as in adult life. Its principle characteristic is that it is limited to the distribution of certain of the cranial nerves. The pain is sometimes supra-orbital; oftener it is situated lower on the face, and is dependent upon the existence of carious teeth.

8. **Uremic Headache.** This form of headache is less frequent than in adult life. It is generally occipital and may occur either in the acute or the chronic stage of nephritis. The presence of other symptoms

of the disease and, especially, the examination of the urine aid in the diagnosis.

9. Migraine (Sick headache; hemicrania).—Headache of this nature has features which are very distinctive. It may occur in children, although much less frequently than later. Neumann[1] reports out of 43 cases 9 beginning in the first 5 years of life; 21 from 6 to 10 years, and 13 from 10 to 15 years. In my own experience it is very uncommon in children. It is often directly hereditary, or may occur in those with an inherited neurotic or gouty tendency. The nature of the cause is not certainly determined. A toxemia has been assumed to be operative, as has also cerebral anemia or cerebral congestion. Females are oftener the subjects of the disease. Some slight cause may start an attack; such as fatigue, indiscretion in diet, emotional disturbance, and the like. The attacks occur more or less periodically at intervals of weeks or months, and appear to be largely independent of the state of the general health. They begin usually with prodromal symptoms of varying nature, such especially as derangements of vision of some sort or vertigo. Very soon there follows a headache of intense severity, generally on one side of the head. This may be accompanied or preceded by various nervous manifestations, among them photophobia, tinnitus, paresthesia in the limbs, vertigo, or temporary difficulty in speech. The face is flushed or pale. The prodromata last a few minutes or longer. The pain continues some hours and is followed by nausea or vomiting; and then by deep sleep from which the patient wakens free from pain. Rachford[2] has seen the attacks of recurrent vomiting in childhood replaced later by migraine.

Diagnosis of Headache.—The diagnosis of headache of any of these sorts is that of the nature of the cause. Localized persistent pain in the head depends oftenest upon organic disease of the brain; that in the course of a certain nerve is neuralgic in nature; temporary headache with fever is most frequently from gastrointestinal disturbance, or occurs with the onset of some febrile disease; periodic headache may be due to migraine; frequently recurring headache may depend upon nervous conditions, anemia, or eye-strain. The position of the pain, too, is often to a certain extent a guide to the cause. Frontal headache is most frequently from eye-strain, disease of the nose, anemia, or gastrointestinal disturbances. Vertical headache is generally of a nervous nature, or dependent upon anemia. Occipital headache is often from eye-strain, otitis, or pharyngitis. Yet only careful, long-continued study will serve to determine the cause in very many instances.

Treatment.—This is necessarily most varied, depending upon the etiology. Iron is required if there is anemia; nitroglycerine if the headache is accompanied by pallor and high arterial tension; strychnine when there is evidence of general debility. Correction of ocular defects should be made. In every case the cause should be diligently sought for and appropriate treatment instituted. Apart from this is the direct relief of the headache at the time. Rest, both bodily and mental; mustard plasters to the back of the neck; hot foot-baths; cold cloths or an ice-bag to the head; the application of the various menthol pencils or headache colognes, are each serviceable in certain cases. The administration of drugs to relieve the pain, such as the coal-tar derivatives carefully employed, may be necessary in some instances. In very frequently recurrent headaches from any source, these, as well as opiates, should be avoided on account of the danger of establishing a habit.

[1] Deutsche Klinik, 1904, VII, 402.

[2] Diseases of Children, 1912, 259.

DISORDERS OF SLEEP

These are of varied nature and may be due to very diverse causes. The following conditions may receive attention:

INSOMNIA. DISTURBED SLEEP

Etiology.—The need of abundant sleep is great in early life. The number of hours which may be fairly allotted as sufficient has already been mentioned (Vol. I, p. 74). During the 1st year the greater part of the twenty-four hours should be passed in sleep. Under many conditions the infant or child is unduly wakeful or the sleep is much disturbed. Among the many frequent causes in infancy is hunger, and this should always be thought of in the case of infants who cry much of the night without evidence of actual disease of any sort. In other instances pain or discomfort of some kind is the agent, usually gastrointestinal, and sometimes from over-feeding. This may be intense enough to cause loud outcries, or only sufficient to produce wakefulness and fretting. In an analogous manner eczema may make a child cry much of the night from the severity of the itching; or pain in some other region of the body, as in the ear, may have the same result. Less intense causes of irritation may be operative, such as a wet diaper, an uncomfortable bed, or the existence of fever. Nervous infants may be very wakeful, either crying during the night or sometimes lying awake in an entirely happy frame of mind. Rickets is a frequent cause of this nervous wakefulness. Many such children are prevented from sleeping by a light in the room or by some unusual noise. A very potent cause is undue excitement before the sleeping hour; as, for instance, when the father returning from business amuses himself by playing with the baby. Still other infants, from undue fatigue or a deferring of the usual time for being put to bed, become too tired to sleep. Rooms which are poorly ventilated or too warm dispose to wakefulness; and either insufficient or too heavy bed coverings may have the same effect. A very important etiological factor is improper training, such as the taking of a child up, walking with it, rocking it, giving the bottle or the breast, and the like, the moment it begins to cry.

After the period of infancy, disturbed sleep is common in nervous, anemic children, or in those with digestive disturbances or suffering from any form of pain. Mental work is a frequent factor during school life, and the existence of fever or of cold feet is also a cause. Especially to be mentioned is the receiving of very decided mental impressions, as from hearing exciting stories or undergoing emotional excitement of any sort, even pleasurable, shortly before going to bed. This may be a prolific source of insomnia or of sleep disturbed by dreaming. Often a nervous child develops an abnormal fear of being alone at night, for reasons which are sometimes not clear to itself; and this naturally produces inability to go to sleep. Disease of the heart or lungs accompanied by dyspnea or cough will prevent sound sleep, and adenoid growths of the nasopharynx may act in a similar manner.

Symptoms.—There may be actual insomnia, or only disturbance of sleep. Sometimes the children lie awake for hours after going to bed instead of quickly falling asleep as a normal child should; but later in the night the sleep is sound. In other cases sleep comes early but the child wakens later and often, and from very slight causes. In still other instances the child, although asleep, is excessively restless and tossing.

Evidently in many cases the sleep is disturbed by dreaming, as shown by the motions of the child, the talking in sleep, or sudden screaming.

Treatment.—This consists first of all in a careful search for the etiological agent, and the removal of this when possible. Nervousness, anemia, debility, and the like, should receive appropriate treatment. Particularly should the nature of the diet and of the digestion be inquired into carefully. Sometimes a lighter evening meal is useful; sometimes the increasing of the amount taken at supper. In infancy the securing an opening of the bowels before the child is put to bed will sometimes be efficacious. In other cases the daily warm bath may be given in the evening with good results. All bad hygienic methods should be abandoned, particularly that of taking the child up on its first cry. The excitement of play before going to bed must be avoided. Sometimes a change in nurses has had excellent results. Quiet at night in a well-ventilated room is important. The room should be darkened; but in the case of older children where fear of the dark has developed, the permitting of a light in the room is advisable if sleep is produced in this way. The amount of mental work in older children should be carefully regulated.

The employment of drugs is to be resorted to only when really necessary, and their use should not be long continued. It is to be noted that the other members of the family are often much more disturbed by a wakeful infant than is the infant itself, who enters upon the next day in good condition while the parents and nurses seem worn out. Drugs should not be given for the sake of these adults. Of sleep-producing drugs to be employed, a choice may be made between the bromides, chloral, veronal, and similar hypnotics. The opiates are indicated only in acute disease, particularly when pain is present.

EXCESSIVE SLEEPINESS

A number of causes may give rise to this condition. In weakly newborn infants there is not infrequently seen a disposition to constant sleep, so great that nourishing the child becomes a problem, the infant falling asleep after only a few sucks at the breast or the bottle. Either debility or a toxemia appears to be the cause. In other cases organic disease of the brain may produce a similar condition. Later in infancy and in childhood excessive sleepiness is seen at the onset of some of the infectious fevers, especially measles; during the course of these diseases or of uremia; if coma is threatening; in organic cerebral disease, especially tuberculous meningitis, either early or late in the attack; after epileptic convulsions; and as a result of drugs, such as the bromides or alcohol, but usually opium given by mothers or nurses generally in the form of soothing syrup of some sort or of paregoric.

DREAMING; NIGHTMARE; SOMNAMBULISM

The tendency in some children to *dreaming* has already been referred to (see above). Even infants may suffer from this, as shown by the sudden startled screaming, or by evidently purposeful movements of the hands. Dreaming seems peculiarly liable to occur in nervous children; in the restless, disturbed sleep of indigestion or over-eating; in respiratory disturbances, as from adenoids; when fever is present; or following some definite excitement or mental strain during the day. Often the dreams are repeated night after night, and when of an unpleasant nature may cause the child to dread going to bed lest they be repeated. The dreams can sometimes be traced to a decided mental impression associated with fear, as when a child has been frightened by a dog, or by the sight of an organ grinder and monkey, or the like.

When such dreams reach an extreme degree, *nightmare* is spoken of. In this the dream is of a most fright-inspiring nature, and of some conditions or surroundings from which the patient is trying in vain to escape. The child awakens frightened, trembling with alarm, but entirely conscious of its surroundings, and able to recall the details of the dream.

In *somnambulism,* the patient, oftenest toward the end of later childhood, performs various systematic actions during sleep, actual walking not being a necessary feature. The causes are the same as for dreaming, but there is a strong individual predisposition; most persons not being somnambulists no matter what the state of the health or the experiences of the day. The patient's eyes are open and the actions systematized, but done entirely without consciousness and without recognition of persons near by. The child may get out of bed, dress, walk or run about, handle books or toys, and the like; and may even perform feats which he could not have done if awake; such as walking safely along the edge of the roof. The dream may be remembered upon the following day, but there is usually no recollection of the acts performed. Occurring alone or often combined with somnambulistic acts in *talking in sleep,* sometimes to such a degree that a long and fairly intelligent conversation is carried on with a person present or imagined to be so. More frequently the talking is not done so connectedly.

The **treatment** of these conditions consists in searching for and removing the cause which occasions them. Children much given to somnambulism may require tying in bed in order to prevent accidents.

PAVOR NOCTURNUS

(Night-Terrors)

Etiology.—The underlying nature of this disorder of childhood is a cerebral disturbance occurring in children with a neurotic inheritance; or with anemia, adenoids, or other cause of general ill health. Exciting influences may be operative, as in ordinary dreaming; among them indigestion, over-loading of the stomach, dyspnea from any cause, mental excitement during the day, and reflex irritation. Yet in very many instances the terrors occur repeatedly without any exciting cause being discoverable. The subjects are oftenest between the ages of 3 and 8 years, but may be older. There is no difference in the predisposition of the sexes.

Symptoms.—Two classes of cases have been described. In the first, denominated *symptomatic night-terror,* the child wakens from sleep perhaps confused, but ultimately cognizant of the dream which disturbed him. The chief cause lies here largely outside of the nervous system itself, the night-terror being a terminal process of an uneasy dreaming sleep, which has preceded it and was produced by extraneous causes. The attack comes on oftenest soon after going to sleep, and is seldom repeated the same night. The condition is so closely allied to nightmare that it is best classified with it, if indeed it is not identical.

The *cerebral* or *idiopathic night-terror,* the one to which the title is properly applied, depends chiefly upon the central nervous disturbance, although exciting causes are operative to some extent. The attack comes on early in the night, after an hour or two of sleep. Without previous warning the child suddenly sits upright in bed bathed in perspiration, screaming, and trembling with terror; or in some instances gets out of bed and is found sitting upon the floor. Sometimes he repeats words or points with his finger, indicating the imaginary object which has frightened him. Although he clutches at his mother or nurse he does not recognize her or know where he is. In spite of the appeals of the mother and the lighting of the room, considerable time is required, perhaps 15 minutes or more, before he can be quieted. Sometimes he sleeps again without returning to consciousness; or in other cases he becomes partially conscious, quiet, and then quickly falls asleep. Urine may be passed involuntarily at the close of the attack; or a desire to be put upon the vessel may be expressed. In contra-distinction to nightmare, the child is unable to describe the occurrence of any dream, does not attain complete consciousness, does not recognize his surroundings, and has no remembrance of the attack upon the following day. As Coutts[1] expresses it, "the child with night-terrors sees visions, while he with nightmare merely dreams dreams." Yet the distinction between nightmare and night-terrors cannot always be sharply drawn.

Course and Prognosis.—The frequency of the attacks varies. They may occur nightly, or only at intervals of weeks or months. Only rarely is there more than one during a night. There are no after-effects seen upon the next day. The prognosis is usually good. It may be months before the recurrences can be prevented, but there seems no certain evidence that pavor nocturnus predisposes to the development later of epilepsy or other nervous affection. That this has seemed possible is due to the fact that some cases of epilepsy exhibit symptoms which closely resemble night-terrors.

Treatment.—Search for the exciting cause should be made and this treated if found. Especial attention should be given to the digestive apparatus. The administration of a gentle purgative at intervals is of service. It is important, too, to see that the evening meal is light. Over-excitement and all nervous over-strain and late hours should be avoided. Yet I recall one instance where putting the boy to bed at a later hour than previously seemed to stop attacks of night-terrors which medication had failed to benefit. Frequently it is well to allow a light to burn late in the room, or to have someone sleep there or in the adjoining room with the door open between. Every care must be taken to improve the general health. Adenoid growths present ought to be removed. The child should spend much of the time out of doors, and the sleeping-room should be well ventilated. Tonics or other remedies are to be given as anemia or evidence of malnutrition or of nervous disturbance demands. In the line of direct medication a full dose of one of the bromides may be administered at bed-time, sometimes in combination with chloral. It is manifest that when the attacks occur at rather long intervals, medication of this sort cannot be continued indefinitely.

PAVOR DIURNUS

Very much less frequently attacks corresponding to pavor nocturnus occur in the day-time during the morning-sleep, or even when the child is awake. As a result of some hallucinations, visual or auditory, the child becomes suddenly terrified, screams, and rushes to someone for protection. There is not entire loss of consciousness of the surroundings.

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[1] Amer. Jour. Med. Sci., 1896, CXI, 156.

The attack lasts a few minutes or may terminate in a paroxysm of crying. The child may be subject to night-terrors also. The prognosis of pavor diurnus is guarded on account of a more possible closer relationship with hysteria, epilepsy, insanity, or other nervous diseases. In quite young children pavor diurnus may be represented by fits of violent screaming without discoverable cause. The element of anger must be excluded in making the diagnosis.

DISORDERS OF SPEECH

Although some of the disorders of speech depend upon organic defects and others are functional in nature, in the following brief description of certain of them they are grouped together as a matter of expediency.

RETARDED ACQUIRING OF SPEECH

The time when children should normally begin to speak has already been discussed (Vol. I, p. 67). Those who, by the age of 2 years, have made little or no effort to talk should be viewed with suspicion, since often this retardation depends upon mental defect. There are many instances, however, where this is not the case, and the assumption of such a cause should consequently not be made too readily. Children who have suffered from prolonged illnesses or who are for any reason physically backward may be slow in acquiring speech. In other instances deafness, more or less complete, is the cause of the failure to learn to talk. The presence of disorders of the mouth, nose and throat is a very exceptional cause. In a considerable number of cases no explanation whatever can be discovered, and the slowness is then but a temporary condition, an individual characteristic, which time will remove. Many of the children who do not talk appear to understand perfectly, and seem merely unwilling to make the effort to speak. Others chatter in a jargon unintelligible to all but the attendants until long after they should have learned to talk clearly. (See p. 283.) In neither case is this a sign of mental deficiency.

The only **treatment** for cases of retarded acquiring of speech consists in continued efforts to teach the child. It is noticeable that those children to whom most attention has been paid, as, for instance, the first-born, usually learn to talk earlier than others. Children indisposed to talk should not have their wants immediately gratified when thay have signified them merely by pointing or other gesture. Otherwise the inducement to make the effort at talking is lacking.

APHASIA; DUMBNESS; MUTISM

In the state of aphasia, taking it in the broader sense, the patient either never develops an ability to use words more than to a very limited extent, if at all; or having had the power, has lost it through accident or disease. In the stricter sense here employed the term *aphasia* is applied only to the loss, through disease of the speech centres or their connecting apparatus, of the power to talk previously acquired; those who never possessed the power of speech or who have lost it through early developing deafness being described as suffering from *dumbness* or *mutism*. In accord with this definition cases of deaf-mutism may be either those congenital in nature, due to a defect of some part of the auditory apparatus; or those where it is acquired through injury or as a result of diseases of the middle or internal ear or of the auditory nerves occurring early in life, especially in the course of the infectious fevers. Dumbness of a greater or less degree is present also in imbecility, depending here upon the mental deficiency, which prevents or delays the acquiring of language. It is seen too in occasional cases where the organs of articulation are defective, and is a temporary condition in instances of retarded acquiring of speech (p. 280).

Aphasia may be either *functional* or *organic.* The first, of a temporary nature, is observed in a number of different conditions. Severe cases of chorea may lose entirely for a time the ability to speak. The disorder is also one of the manifestations of hysteria; has occurred during an attack of migraine, or has followed a shock of some sort. Not infrequently in children a temporary aphasia follows a severe attack of an infectious fever, especially typhoid. Organic aphasia results from a variety of causes, among them trauma; hereditary syphilis; cerebral paralysis; tumor of the brain; encephalitis, etc.

Only exceptionally is aphasia sensory in type, the patient being unable to understand words although he can speak more or less clearly. Nearly always the aphasia of children is motor, the ability to enunciate being absent, although the understanding of spoken words remains. Aphasia may be complete, or the child can speak some syllables or words.

The **prognosis** depends upon the cause. In the functional cases of aphasia it is entirely good, and even in those due to organic causes the frequency in early life of the regaining of the power of speech is surprising. In the partial mutism seen in imbecile children, the extent to which the child will learn to talk depends upon the degree of mental defect and the efforts put forth in the way of instruction. The only *treatment* possible in any case of aphasia is that of education, combined with efforts to improve the general health.

Alexia (*Congenital Word Blindness*).—This is an inability or retardation in acquiring the power to recognize written or printed words or letters; while a few patients seem to recognize them but cannot put them into speech. The child is intelligent, the vision unaffected, and the understanding of spoken words and the ability to talk is unimpaired. The symptoms vary from complete incapability of learning to read, down to a mere slowness in acquiring the power, which may be wrongly attributed to stupidity or defective eye-sight.

STAMMERING; STUTTERING

These two terms are very commonly used interchangeably and synonymously; and when a differentiation is attempted the distinguishing features are not very sharply and uniformly defined by writers. Thus divided, *stuttering* is applied to a difficulty in beginning a word or syllable, often with frequent repetition of the first portion of it; while *stammering* denotes trouble in uttering certain sounds at all, is not accompanied by repetition, and is in some cases associated with malformation of the organs of speech or with imperfect intellectual development. Many include also *lisping* and *lalling* (p. 283) under stammering. We may conveniently with Wyllie[1] regard the two main forms as indentical and speak of the condition as *stammering:* "stuttering" being merely a variety in which there is a rapid repetition of the difficult initial sound. German writers appear to prefer to "stammering" the equivalent of the English term "stuttering."

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[1] The Diseases of Speech, 1894.

Etiology.—The condition is a common one. Gutzman[1] found that in 155,000 children in the Berlin public schools there were 1500 stammerers; *i.e.* 1 per cent., and Priestley[2] in a study of about 20,000 in England, estimated it at 0.4 per cent. The underlying cause is a lack of nervous coördinating control over the respiratory and the articulating mechanisms. To speak it is necessary that there shall work in unison the expiratory apparatus, the muscles of the larynx, and the nerves controlling the lips, tongue and palate. Defect in the action of any one of these may produce stammering. Most frequently it is the control of the expiration which is at fault. Often the action of the brain is too fast for the articulatory apparatus, and the coördinating control is lost under the excitement.

Heredity plays an important rôle. Gutzman[3] found that in 28.6 per cent. of 569 affected children stammering existed in other members of the family also. Yet many of these cases apparently of inheritance are doubtless dependent upon unconscious mimicry. Many of the subjects are of a neuropathic disposition and may exhibit other nervous disturbances. Disease of the respiratory tract is present in a large number. The male sex is distinctly more disposed (3:1). As regards the age, the affection may appear under that of 4 years, but is less common then and more liable to be temporary or occasional; recurring with any disturbance of the general health or some emotional strain, and disappearing with the removal of the cause. The instances of persistent stammering develop usually in later childhood. Among influences which may be considered the exciting cause of persistent stammering, or which make it worse if already present, may be mentioned excitement; impairment of the general health; digestive disorders; and disturbances in the respiratory tract, such as adenoids.

Symptoms.—The principal symptom is produced by a spasm of the articulating apparatus which prevents for a moment the word being spoken, or breaks in upon it during its utterance. Various grades may be observed. In the milder the stammering is only moderate and at times entirely absent; but reappears when the patient is excited or hurried. Often only certain sounds cause the chief hesitation and difficulty, the words which immediately follow being often hurried through rapidly. There may be a complete momentary stopping over the difficult word or syllable, with a final explosive enunciation of it; or the sound may be repeated a number of times in rapid succession before the rest of the syllable can be pronounced. In the severer cases various movements may accompany the effort at articulation; such as contortion of the face, or certain systematic motions of the arms and legs; while in some instances there is the interjection of grunting or other noises, or of words without bearing upon the sentence. It is a curious fact that many stammerers can whisper without the slightest difficulty, and most of them have no trouble in singing.

Prognosis.—In the stammering beginning in children less than 4 years of age, the prognosis is on the whole good. The condition is then usually less severe and only temporary, and soon disappears permanently if attention is paid to the improvement of the general health and to care in training to talk properly. In older children the prognosis is much more uncertain, and the longer the disease has continued untreated, the less likely is it to be cured. Relapses are very liable to occur unless treatment is persisted in.

[1] Das Stottern, 1898, 329.

[2] Brit. Journ., Child. Dis., 1916, XIII, 104.

[3] Grancher and Comby, Traité des mal. de l'enf., 1905, IV, 209.

Treatment.—As so many of the patients are neuropathic, great attention must be given to every means which will diminish the tendency to nervous disorder and will improve the general health. The diet and the exercise should be carefully ordered. Separation from stammering associates is of vital importance, as when several in a family suffer from this disease. For the same reason removal of a stammerer from school is advisable, both for his own sake and for that of other children; since it is particularly during school-life and under school-influence that stammering develops or is liable to grow worse if already present. Inasmuch as it is usually under excitement that the little child is most liable to begin stammering, in his attempts to talk with undue rapidity, his efforts at conversation of this sort should be stopped at once, and he should be made to tell slowly, in a low voice, and with short sentences what he has to say. This early management is of great value in preventing the habit from becoming fixed.

For the direct treatment of stammering already established in older children, daily exercise in breathing and speaking should be carried out. The underlying principles of this consist in deep respiration; speaking with the lungs full of air; exercises in singing, whispering, and then in faintly spoken words; reading out loud in a monotone; and the like. But satisfactory results can be obtained only by long persistence, and under the training of a teacher especially accustomed to this work. The methods are carefully described in the works of Gutzman,[1] Wyllie[2] and others.

LISPING

(Lalling; Alalia; Idioglossia)

Lisping is an imperfect articulation, in which certain letter-sounds are replaced by others, the most frequent being that of "th" for "s"; but the defect is by no means necessarily limited to this, the letters "r," "k," "g" and others being mispronounced in some cases. Such replacement is a constant normal attendant of the early efforts of a child to acquire speech, and is due to an improper position of the organs of articulation in the forming of words. Ordinarily the difficulty rapidly disappears as the child learns to talk, but in some instances it continues to a greater or less extent and is then pathological. In some of the severer cases there may be an organic defect present, such as harelip, cleft palate, paralysis of the lips or palate, etc.; but this is not true of the majority.

The worst form of lisping is sometimes spoken of as *lalling*, and consists of a persistence of infantile speech. It is observed oftenest in children with mental deficiency. In an exaggerated condition there is *alalia* or an entire inability to articulate. The term *idioglossia* has been applied by Hale White and Golding Bird[3] to an aggravated form of lalling, in which there is so much substitution of one sound for another less easy to the patient that, although the child is mentally bright, the speech is voluble but is practically unintelligible. The condition dates from the time talking first began.

The **prognosis** of lisping is good in all functional cases if treatment is carried out properly. The **treatment** consists in education by mechanical measures and the like, as in the oral methods with the deaf mutes by aid of the touch and the sight, with the purpose of bringing the organs of articulation into the proper position. This can be carried out effectively only by a trained teacher. Organic defects require suitable operative measures.

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[1] *Loc. cit.*
[2] *Loc. cit.*
[3] Med.-Chir. Transac., 1891, LXXIV, 181.

NEUROTIC HABITS

These are numerous in infancy and childhood and of great variety. Some of the more important may be considered here. In many instances they are matters of little moment, while in others they appear to be manifestations of degeneration, are to be classed among evidences of minor psychic disturbances, or are well-marked in the true psychoses.

THUMB-SUCKING, TEETH-GRINDING, NAIL-BITING, ETC.

Thumb Sucking; Tongue Sucking.—Although the predominance of cases in which the thumb is the object sucked gives the name to the habit, other parts of the body may be the portions thus treated, as the fingers, toes, tongue, lips, etc.; or some other object may be used in this way, as a rubber nipple, the corner of the blanket or sheet, or a part of the clothing. The sucking of the tongue is a common symptom in Mongolian idiocy. Thumb-sucking is exceedingly common in infancy, and is usually a matter of little importance at this time. Few pediatrists would be willing to accept the view advanced that it has any sexual basis. It arises primarily from the natural disposition of the hungry infant to put something into his mouth. Generally it is "simple" in character, according to the nomenclature employed by Lindner[1] the child doing only this. In the "combined" cases the sucking is accompanied by other acts, such as rubbing at or boring into the nose, rubbing the ear or the genitals, pulling at the hair or the navel, and the like. Thumb-sucking is more liable to occur when the child is about to go to sleep, or when hungry, sleepless, excited, or not feeling well. In the milder cases it is then only occasional, and the child is easily diverted from it. Under these conditions, and when clearly not increasing in frequency, it may be ignored until the 2d year is reached. If it is very continuous, efforts should be made to stop it promptly, on account of the danger of its producing permanent deformity of the thumb or of the jaws, with malposition of the teeth.

The **duration** of the habit is indefinite. Most infants stop it of their own accord, but in other cases, especially where there is a neuropathic disposition or a tendency to mental weakness, the habit may last into childhood. The ultimate **prognosis** is usually good, but recovery often tedious if treatment has been long deferred. Children who seem to have been cured readily relapse if the general health suffers in any way. Only in decided cases of mental deficiency is the final cure uncertain.

All that has been said about thumb-sucking applies, of course, equally well to the sucking habit, however, shown. The sucking at a rubber nipple or "comforter" should only be permitted under certain not frequent conditions to quiet unusual nervousness or its attending symptoms.

Treatment should be commenced as early as possible if the thumb-sucking is unduly frequent, and in all infants over a year of age. In the early stage mechanical restraint is the most effectual method. An elbow-splint or a pasteboard cuff may be put upon the elbows, or mittens or other hand-covering used, or the sleeve of the dress pinned to the bed. Very satisfactory globular aluminum mits are obtainable. (See p. 562.) The application of bitter substances to the thumbs, such as a solution of aloes or of quinine, is of service only in the mildest cases. Mechanical interference with tongue-sucking or lip-sucking is hardly practicable; but the habit generally ceases of its own accord as infancy is passed.

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[1] Jahrb. f. Kinderheilk., 1879, XIV, 68.

In older children continued mechanical restraint and constant supervision are necessary to obtain a cure, but threats and punishment are useless and often drive the child to a surreptitious practice of the habit. Rewards are of more value; and the awakening of a sense of shame and the engaging of the coöperation of the child are often efficacious. Cure is sometimes accomplished by the ridicule of playmates. Inasmuch as after the period of infancy the habit is a distinct neurosis, all causes for ill-health and nervousness must be diligently sought for and removed.

Tongue-Chewing.—In this nervous habit, occasionally seen, one side of the tongue is subjected to a chewing movement between the molars. It begins oftenest in the 2d year of life. The movement is rapid, and the tongue is made red and inflammed. The habit may persist throughout life.

Teeth-Grinding.—It is only in a very limited sense that this can be considered a habit, since it is observed chiefly during sleep, or in unconscious states dependent upon disease. It occurs with greatest frequency in neurotic children suffering from some reflex peripheral irritation, and is consequently common in gastrointestinal disorders of different sorts, but is in no way especially indicative of the presence of worms. It is also the result of direct cerebral irritation, and hence is a characteristic symptom of meningitis. The frequency with which it develops and its significance in cases dependent upon peripheral irritation vary greatly; some children exhibiting it on the occurrence of the slightest disturbance of this sort; others never.

Teeth-grinding is not infrequently seen in imbeciles during the waking hours, and sometimes appears to be a vicious habit even in those not thus mentally affected. In bad cases the teeth become much worn. **Treatment** must be directed against the cause, including both the general condition and any peripheral irritation present.

Nail-Biting.—Biting of the nails is observed only in decidedly nervous children, and may persist more or less during life unless vigorously treated. It is of much greater significance of a disordered state of the nervous system than is thumb-sucking. It may be done every day, or only at times when the general nervous condition is worse, or the patient is under some sort of excitement or worry. **Treatment** is the same as for thumb sucking. The nails should be kept cut very short, and the fingers protected by gloves. In addition, measures should be employed for the relief of the inherent nervous condition.

Picking.—Picking, pulling, rubbing, or scratching at some portion of the body is seen in infants and children of nervous disposition, or those affected by some disorder of general nutrition, such as rickets. To this is often added a localized irritation. There may be an itching cutaneous area without visible eruption which accounts for the act; but in very many cases no such condition is discoverable and the habit is purely a nervous expression. Thus children suffering from illnesses of many sorts, such as typhoid fever, often pick at the lips so continuously that they require restraint. Fretful infants frequently pull at the hair or the ears. Certain infants have a tendency to pull at the foreskin, the ear, the navel or other parts of the body while sucking the thumb. The most frequent and annoying habit, however, is that of picking at or boring into the nose; a habit unfortunately by no means confined to early life. Sometimes there is distinct local irritation causing this, and the violent rubbing at the nose often done by infants may be occasioned in this way. In other cases, however, it is purely a nervous habit, often most marked when other nervous symptoms are increased in severity.

In the way of **treatment** for picking of any sort, forcible restraint may be necessary in infancy; and admonition and restraining measures of other nature in older subjects. Of especial importance is it to improve the general nervous condition and to remove the local irritation.

RHYTHMICAL MOVEMENTS

(Head-Banging; Swaying; Head-Nodding; Head-Rocking, etc.)

In some children rhythmic movements of various sorts may be observed. Certain of these occur oftenest in those mentally defective, but others may be seen in subjects merely of a nervous disposition or even apparently entirely normal in this respect. The movements may be always of one sort, or one form may alternate with another.

Head-banging, as it was named by Gee,[1] is encountered in extremely nervous subjects often suffering from rickets, or in those mentally defective. It occurs in children generally of from 2 to 6 years of age, and while awake or sometimes in sleep. The child rhythmically bangs his head against the pillow or the side of the crib and may continue this for minutes or hours. Sometimes there is evidence of anger; generally none at all. The force used may be sufficient to bruise the skin.

Head-rolling is not infrequent in infants, especially as a manifestation of a nervous disturbance oftenest associated with rickets. The child lying in bed rolls its head from side to side very frequently during the day, and continues this day after day. As a result the hair is almost completely worn away from the back of the head. Zappert[2] has described a nocturnal rhythmical rocking movement occuring only during deep sleep, and seen in later childhood. He regards it as similar in nature to nervous habits observed during the waking hours.

Head-nodding is another of the nervous movements sometimes seen. It is to be distinguished from the gyrospasm and nodding spasm previously described (p. 266), in that it is much more energetic and apparently intentional.

Swaying, or **body-rocking** to and fro is a common habit, especially, but not only, observed in mentally defective children. The child in a sitting position sways forward and backward rapidly and continues this for hours uninterruptedly. In some cases this may perhaps be allied to the buttock-rubbing which is a form of masturbation in infants; but generally it is too continuous and without the evidence of excitement seen in that condition.

The **treatment** of these various conditions consists in improving the general health, combating any nervous condition present, and in forcible restraint as far as possible.

PICA

Pica, or perverted appetite, is a common disorder observed oftenest in the 1st and 2d years of life, although sometimes developing later; and consisting in the desire to eat a large variety of unsuitable substnces. Often there is an underlying digestive disturbance, while in other cases the presence of intestinal parasites, especially hook-worm, is a cause. In most instances the child seems in good general health; in others there is anemia, malnutrition, or a distinctly neurotic disposition. The habit is frequent in mentally defective subjects.

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[1] St. Barthol. Hosp. Rep., 1886, XXII, 97.

[2] Jahrb. f. Kinderheilk., 1905, LXII, 70.

The normal infant soon learns by trial that only certain articles he picks up are good to eat. The child with pica, on the other hand, seems unable to acquire this knowledge, and even develops an unnatural craving for inedible articles, such as sand, earth, wool from the blankets, hair from his head, coal, ashes, plaster from the wall, etc. Sometimes only one improper article is taken; in other cases many sorts. The *course* of the disease is often prolonged unless early efforts are made to check it. There is a natural tendency for pica developing in infancy to cease of itself at the age of 3 or 4 years, and this renders the *prognosis* good; but the habit may continue much longer and be very intractable. The general health may be seriously affected by the harmful action of the various objects swallowed, and the loss of normal appetite which commonly attends. *Treatment* should be given promptly, and consists principally in preventing the eating of the unnatural articles. If this is commenced early, recovery usually soon follows. The general health and especially the digestive apparatus must receive appropriate treatment. Change of air and scene is often beneficial.

HOLDING THE BREATH

There is a certain apparent relationship in the symptoms between attacks of this nature and those of laryngismus stridulus. True cases of the latter disease are dependent upon the spasmophilic diathesis and exhibit other symptoms of this condition. (See p. 256.) The attacks of holding the breath, as now under consideration, are to be classified as the direct result of yielding to violent anger or to other excitement, operative in a highly neurotic uncontrollable child; and, indeed, are of a psychopathic nature. A temporary spasm of the larynx is probably produced. They occur at a somewhat later period of life than laryngismus stridulus, exhibit no evidences of spasmophilia, and appear to possess no relationship to epilepsy. The holding the breath cannot in itself be called a bad habit, although it seems sometimes to be voluntary; but is the result of the lack on the part of the child of the exercise of any control whatever over its emotions or impulses. The attacks begin oftenest in the 2d or 3d year of life, but generally disappear by the age of 4 or 5 years at latest. Although anger is the most frequent cause, in some cases sudden fright is operative.

Symptoms.—Under the influence of something which displeases or startles him, the child exhibits a sudden cessation of respiration, cyanosis, and rigidity of the body; and then in severe cases loss of consciousness, possibly convulsive twitching, pallor which is sometimes extreme, and general relaxation. In a few seconds the attack is over, and in a very brief time the patient seems as well as before. In some cases the holding of the breath is the terminal stage of an attack of rage in which the child throws himself on the floor and screams violently for a few moments. In others violent crying succeeds the initial cessation of respiration, or the holding of the breath is both preceded and followed by crying. The attacks may occur several times a day or at much longer intervals. The **prognosis** is good and, unlike laryngismus, the condition is never a fatal one, and under the influence of appropriate treatment recovery will gradually take place.

Treatment.—This consists, first, in measures directed to improve the nervous condition which is the primary cause. Life should be largely in the open air; anemia, rickets and other debilitating diseases relieved; and all exciting causes avoided. Especially important is the teaching of the child to exercise his will-power in the control of his passion. Rewards and encouragement may do much; and certainly the yielding by the parents on every occasion for fear of precipitating an attack can do only harm. Whether or not punishment shall be administered depends upon the individual case, since the punishment itself is liable to precipitate an attack in some instances. Yet warning in advance that punishment, not corporeal, will follow, may aid the child in learning control. Certainly nothing must be done to frighten the patient, and kindness will do more than harshness with the neurotic condition which these children possess. The attack itself may sometimes be curtailed by the sudden application of cold water or a cold cloth to the face; yet this is seldom required and is not well tolerated in every instance.

MASTURBATION

The frequency of this neurotic habit is enormous and usually underestimated. It may begin in infancy, even in the 1st year of life, and is then more common in females and exhibited in the condition denominated "thigh friction." This is not infrequently observed in the practice of every pediatrist. At this time of life it can scarcely be called strictly a sexual expression, although the symptoms are undoubtedly those indicating the production of something which at least simulates an orgasm. The act at this age has been called by Rachford[1] "pseudomasturbation." After this period, particularly in later childhood, there is an undoubted sexual pleasure derived from the habit, and the title "masturbation" is unquestionably appropriate.

Etiology.—A marked neurotic disposition is an undoubted powerful predisposing cause, and it is only in such cases that the habit becomes a matter of serious moment. In many imbeciles, or in those who later show themselves vicious in other respects, masturbation has often begun early and is practised to great excess. The general health, too, is another factor, and anything which tends to its impairment may predispose to masturbation or cause its renewal if temporary cessation has occurred. Some local irritation is active in many instances, such as the presence of phimosis, the accumulation of smegma behind the corona, balanoposthitis, the irritation from tight diapers or drawers, vulvovaginitis, the itching produced by eczema or by the presence of thread-worms in the rectum or vagina, constipation, highly acid urine, preputial adhesions of the clitoris, and the like. Sometimes irresponsible nurses have habitually rubbed the penis of an infant for the purpose of soothing him. In older children it may have been learned accidentally, as by sliding down a balluster rail, climbing trees, horseback riding, irritation of tight clothing, and the like; but oftenest at this age it is taught by companions. It is now much oftener practised by boys, although as puberty is approached it is frequently encountered in girls as well. It is much more common in later than in early childhood.

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[1] Arch. of Ped., 1907, XXIV, 561.

Symptoms.—In infancy masturbation shows itself usually in the form of thigh-friction or associated movements. The infant, usually a female, sitting on the floor or lying on its back with the lower extremities drawn upward, rubs the thighs vigorously together, thus catching and rubbing the clitoris between them. The face meanwhile becomes flushed and the eyes have a fixed, somewhat staring expression and take no notice of surrounding objects. Often the attention of the attendants will be drawn by the unusual absence of noise on the part of the child. After a short time something suggesting an orgasm is produced, sweat breaking out on the forehead, the face growing paler, and the infant lying back relaxed from temporary exhaustion. The procedure is repeated possibly many times a day or even during sleep.

Various modifications or substitutions of this typical thigh-friction are seen. Some infants lying on the back rub the buttocks from side to side on the bed or floor; others rub the genital organs against some object, such as a pillow, the leg of a chair, or the like. Sometimes movements of other parts of the body are associated with or even appear to take the place of those which directly cause friction of the genitals.

Infants with thigh-friction may exhibit other signs of neurotic excitability or show evidences of retardation. Many such manifestations present are, however, less often the result than the cause of the habit. Many infants seem perfectly healthy. The examination of the genitals will frequently show redness of the entrance of the vagina, and probably some local source of irritation.

After the period of infancy, masturbation is more clearly associated with an early development of sexual excitability. The hand is generally used, thigh-friction being uncommon. As a result of reproof and consequent consciousness of wrongdoing, the habit soon becomes a secret one. Generally it is occasional only, but in bad cases in children of a very highly neurotic nature the child may lie awake at night masturbating, or do it many times in the day, and sometimes loses all sense of shame and power of self-control, and masturbates in public as well. The local manifestations are varied. The penis may be unusually turgid or enlarged, and with a tendency to ready erection, or the organ may seem relaxed. The prepuce may be swollen and slightly inflamed. In girls the clitoris and labia may be larger than normal and reddened, and there may be some degree of vaginitis. None of these signs, however, are positive, and all may be wanting. As regards the general symptoms there is much uncertainty. In many cases there are none discoverable. Excitability, nervousness, apathy, mental hebitude, depression, morbidness, shyness, reticence, pallor, loss of memory, debility, headache and many similar conditions have often been described as the result of masturbation; but it seems more probable that, in the majority of instances, these are manifestations of the neurotic state upon which the habit itself largely depends. Feeblemindedness or other psychic disturbance, for instance, is far oftener the cause of masturbation than its sequel; and the same is true of hysteria, epilepsy, nymphomania, insanity, sexual perversity, and the like. These conditions were developing, and excessive masturbation was but one of the early manifestations. On the other hand, frequent masturbation certainly has an exhausting effect upon the nervous system, and may make worse the symptoms of general nervous debility already present; and the consciousness of concealed wrongdoing is harmful to the child's moral nature.

Prognosis.—This is good in the case of infants with thigh-friction. Treatment is generally successful, and even without this there is a disposition for the habit to cease of itself as early childhood is approached. Moreover, there is usually discoverable a cause which can be removed. Occurring in early childhood, the prognosis is as a rule good. There is a tendency to recover from this as from any other habit-neurosis. An exception exists in those cases where the masturbation is very frequent and uncontrolled by the patient, and where there is a distinct pathological psychic condition shown in other ways. In older children the prognosis is much more uncertain, since the habit readily becomes a fixed one, and as it is carried on without the knowledge of the parents, supervision and control become a matter of great difficulty. The prognosis, however, of the general condition of the patient is good, except in the cases where the masturbation is excessive and uncontrollable, and is often performed in public. Here it is frequently an evidence of degeneration and but little can be done for it. It is remarkable, as pointed out by Neter[1] how little damage seems to result in some instances, even in the case of masturbation in young children carried on to an excessive degree.

Diagnosis.—This can be made with certainty only by the observation of the child during the act. I have known the greatest alarm and mortification aroused in the minds of parents by the injudicious statement of a nurse that a child was a masturbator, founded on nothing but her preconceived idea, without any facts whatever.

Treatment.—In cases of thigh-friction careful constant supervision by the parents is important, and the act must be interrupted as soon as it begins by picking the child up and diverting its thoughts into other channels. In bad cases, and especially if the act occurs during sleep, some appliance must be employed which will mechanically make friction impossible. A small pillow may be placed between the thighs and a bandage applied around them; or the knees kept separate by a rod terminating at each end in a leather collar fastened around the thigh just above the knee. In infants or quite young children where the hands are employed, it may be necessary to confine these, as by elbow-splints, or by tying them in some way. In cases of any age, search must be made for local causes and these removed. Adhesions of the prepuce should be separated, and in any case cleanliness of the penis insisted upon. Circumcision is of value if there is a narrow prepuce. It is also sometimes curative in older children through the soreness produced by the operation, and the consequent breaking in upon the habit. Adhesions of the preputial hood in females should be freed, and if necessary circumcision of the clitoris performed. Vulvovaginitis, if present, demands treatment, as do thread-worms, constipation, and eczema. Guarding against tight clothing is importamt.

Further treatment in the case of older children is difficult and varied, must often be long-continued, and is frequently unsatisfactory. The general health must be looked after in most instances, and hygienic measures employed to control nervous excitability. A life in the open air with abundance of exercise is greatly to be desired; avoiding climbing trees, horseback riding, and the like, which might cause local stimulation. No reading should be permitted which excites the nervous system in any way; and, in fact, much use of books of any sort is to be deprecated. Sleeping in very warm soft beds under warm bed-clothing is to be avoided. Cool morning baths have a beneficial effect. The meals should not be too large or too stimulating, and constipation is to be prevented. The child must be kept from vicious companions, and, conversely, his associations must be so controlled that he cannot teach the practice to others. A constant supervision must be kept over the patient, making this as complete as possible, so that few opportunities are given to be alone. Yet in all this, as well as in the matter of giving advice and warning, the most difficult problem is to do enough and yet not too much. Punishments are of little value, and only make the patient more secretive. Dwelling upon the matter as a sin may breed in nervous children only despair and cessation of effort, if good resolutions are broken. Such warnings of the terrible results as appear in the quack-medicine advertisements have rather a bad than a good effect on neurotic subjects. The confidence of the child should be sought, and encouragement given; and the practice put before him as one which will make him inferior to other boys in the matter of strength of body, and of a less manly nature. Meanwhile, great caution is to be observed lest even such advice is too often given; and surveillance which is too apparent serve only to concentrate the mind of the patient upon his practices and to establish the habit more firmly. All methods should be such as tend to keep the child's mind away from himself.

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[1] Arch. f. Kinderh., 1913, LX–LXI, 497.

CHAPTER III

PSYCHIC DISORDERS. PSYCHOSES

The psychic disorders are in a way a connecting link between the functional and the organic nervous diseases, inasmuch as, although all of them exhibit disturbances of function, in some, at least, these rest upon a distinct anatomical basis, and are often associated with the stigmata of degeneration described later. (See p. 296.) The range of subjects included varies according to the classification of different writers. By many hysteria is placed in this category, and even though not a psychosis, it is at least a disturbed psychic state. So, too, many of the symptoms already detailed as occurring in nervous children are properly viewed as psychic disturbances. The following may be considered in this connection:

HYSTERIA

Although the symptoms of hysteria are manifold, and many shade imperceptibly into those of neurasthenia, various neuroses and psychoses, and organic diseases, the malady in itself is entirely distinct from other affections. Its underlying character is a lack of will-power, with emotional and reflex excitability; combined with the production of a great variety of symptoms apparently due to corporeal affections, but in reality simulative and of a psychic nature. There exist fixed or perverted ideas which are often produced by suggestion or autosuggestion. The malady is an entirely psychic one. There is an exalted egoism, and a desire to attract attention and gain the sympathies of others; and the power of self-control is enormously decreased.

Etiology.—An affection principally of later years, nevertheless, the occurrence in some of its forms in childhood is certainly more common than often supposed, and there is reason to believe that it may be observed even in infancy. Heredity is a prominent *predisposing factor*, and the tendency to hysteria is a congenital condition; although the manifestations of it are usually first seen much later. Children born of parents with nervous diseases, or themselves of a neurotic nature, are particularly likely to develop hysterical symptoms. The predisposition is greater in females, but not with the preponderance seen in adult life. The more nearly puberty is approached, the more frequent the disease, and the greater proportion of females attacked.

Yet the development of the hysterical tendency generally antedates puberty, although perhaps long lying dormant, and may depend upon the forgotten suppression of some emotion, or the impression made by some incident experienced early in childhood, as claimed by Freud and his followers. The theory that these are always of a sexual character is, I believe, entirely unproved, or that all cases of hysteria in children are the result of earlier forgotten repressed emotions of any sort. The neurotic disposition upon which hysteria rests is, as stated, congenital, but the production of symptoms depends upon a variety of influences.

Among the *direct causes* of the development of hysteria are malnutrition of any sort; surroundings favorable to emotional excitement; the strain of school life; imitation from association with hysterical or other nervous individuals; faulty training by parents; the incurring of fright, fear, or trauma; the impairment of health following acute diseases; and sometimes masturbation or other genital irritation.

Symptoms.—These may be of the most varied form, and may simulate disease of any part of the body. They may come on suddenly and disappear rapidly, or may be very persistent. In contrast to the condition observed in adults, very commonly there is at first in early life only a single symptom prominently manifest (monosymptomatic); and the stigmata of hysteria are more often absent until puberty is approached, when suggestion may develop some of these, especially during examinations. This fact depends upon the simpler methods of thought of the young. Among the stigmata may be mentioned anesthesia and hyperesthesia of different sorts; the globus hystericus; contraction of the visual fields; ovarian and spinal tenderness; and various convulsive conditions. The general health of patients with hysteria is usually not perfect. Disorders of digestion, disturbances of appetite, anemia, insomnia, and intellectual derangements are common.

Only some of the more important symptoms can be considered; conveniently classified as sensory; motor; psychic; and those connected with the internal organs and the special senses.

Sensory Symptoms.—These constitute a very common manifestation of hysteria in early life. Prominent here is hysterical headache, which appears especially when it is of service to the child. Neuralgic pain in various regions may be hysterical, as may sometimes pain in the epigastrium, or in the region of the navel or of the appendix. Pain with contracture about the joints is one of the most frequent manifestations in children and may last for months. Sometimes there is general widespread hyperesthesia with suffering produced by movement, entirely prohibiting any passive or voluntary motion. I have seen the latter in a boy of 11 years who, however, could and did move his limbs without the slightest difficulty during sleep. Hyperesthetic or anesthetic areas may sometimes be found, but not as frequently in children as later in life. Sometimes there is paresthesia, such as a feeling of coldness or deadness in some region. The disturbance of sensation is not in accord with any distribution of the nerve trunks. It may be discovered on the surface of the body, in the pharynx, or on the conjunctiva; or there may be pain and tenderness in small areas in the region of the spine or over the ovaries. Not infrequently the sensory symptoms directly follow an injury, and may then be very confusing in making a diagnosis.

Motor Symptoms.—Manifestations of this kind are of great variety. The more common condition is *spasm* of some sort, either tonic or clonic. In the most severe cases there occurs a typical hysterical convulsion, frequently with opisthotonos; which, with all its violence, has no attendant biting of the tongue, injury from falling, incontinence of urine, or true unconsciousness. The general hysterical convulsion is seen usually only in older children, but not always so. I have known the diagnosis from epilepsy only possible through the distinct remembrance by the patient of the nature of the occurrences during the seizure; and recall another case, a girl of 9 years, who suffered from mild convulsive attacks, which ceased to appear immediately on removal from the anxious sympathy of the mother. Catalepsy may sometimes occur, and is of a hysterical nature. In milder cases more limited tonic or clonic spasms of various parts of the body may be present. These may not infrequently be of a choreiform nature. In others the spasm is represented by contortions of the face; irregular action of the diaphragm and other muscles of respiration; torticollis, or hysterical contraction elsewhere. Some of the cases of tic and of chorea electrica are probably hysterical.

Paralysis may develop, with or without contracture. This may be flaccid or spastic; paraplegic, hemiplegic, or monoplegic; or affect only one portion of a limb. It is less common in children than are the evidences of spasm. Astasia abasia is one of the commonest forms of hysterical paralysis in children; the child being able to make all or many of the normal movements with the limbs, yet unable to stand or walk, or doing it with difficulty. Aphonia may occur from hysterical paralysis of the muscles of the larynx. The tendon-*reflexes* in hysteria are usually active; never absent. The cutaneous reflexes are variable; those of the cornea and pharynx perhaps being absent. Ankle-clonus does not occur. *Tremor* is an uncommon symptom.

Psychical Symptoms.—Here are to be classed attacks of laughing or crying without sufficient cause; violent paroxysms of anger; great excitability of the nervous system; disturbed sleep with night terrors or somnambulism; capriciousness of disposition; unreasonableness; impulsiveness; hysterical aphasia; a stuporous condition; delirious states sometimes of a maniacal nature; hallucinations. Not all of these, of course, are necessarily hysterical. An extreme power of imagination, irritability, excitability, egoism, and a remarkable tendency to deception without reason, are characteristic psychic alterations. It is to be noted that stigmata of degeneration, as seen in the psychopathic child, or even actual psychoses, may be combined with or follow upon hysteria. These are not, however, a part of the disease.

Symptoms Connected with the Special Senses.—In this category are the manifestations encountered in connection with the eye and ear. Blindness is an unusual symptom. Contraction of the visual fields is very uncommon in children. Photophobia is frequent, as are forms of asthenopia. Blepharospasm with closure of both eyes may occur, or there may be paralysis of some of the ocular muscles. Hysterical deafness is not uncommon, following perhaps a blow on the ear.

Symptoms Connected with Other Organs.—These symptoms, as with hysterical manifestations of any sort, may be the result of autosuggestion, dependent upon the occurrence earlier of diseases connected with certain of the organs. Thus a spasmodic cough, or the development of hysterical vomiting, may be properly assigned to the influence of previous cough or vomiting which had been produced by actual disease. *Respiratory symptoms* may be represented by sighing or rapid respiration; yawning; hysterical cough; attacks of dyspnea; rapid respiration; aphasia from the paralysis of the laryngeal muscles, as already mentioned; hiccough from spasmodic action of the diaphragm. Of these cough is one of the most frequent and troublesome. Among *circulatory symptoms*, are palpitation; tachycardia; pallor; flushing, and edema. The disturbances connected with the *digestive organs* are of much interest. Hysterical dysphasia may occur from spasm of the esophagus. In one instance in a boy of 8 years under my observation, the condition was believed to depend upon organic stenosis, until under anesthesia sounds of the proper size were found to pass without difficulty. Gastralgia may be encountered. Vomiting, often of a very persistent nature, is a frequent symptom. Even in infants may be observed vomiting which follows in some cases the administration of any food of which the taste is new or not entirely agreeable; and the ability of many older children to vomit at will must be put in the category of hysterical manifestations. Anorexia nervosa (Vol. I, p. 706) is a characteristic hysterical disturbance; and I have observed it even in infants in the 1st year of life. Among other symptoms connected with the alimentary tract, which may sometimes be of a hysterical nature, are meteorism; long-continued diarrhea; constipation; incontinence of feces; and recurring anal prolapse. Of *genito-urinary* manifestations are especially to be mentioned frequent urination and pollakiuria, both of which are common. Ischuria and paralysis of the bladder are less frequent. That enuresis nocturna is sometimes amenable to treatment by suggestion places such instances among urinary manifestations of hysteria.

Prognosis.—The prognosis is, as a rule, good if treatment is instituted early; yet this statement applies only to the cure of the visible conditions present. When the disease appears in a child of a distinctly neurotic tendency or inheritance, return of hysterical manifestations is very liable to occur in later years. Consequently the prognosis for ultimate complete recovery is often most uncertain, although decidedly better than in adult life. The suddenly developing attacks, if properly treated, offer the best prognosis, and this is especially true of cases where imitation has been an active cause.

Diagnosis.—The principles upon which the diagnosis rests have already been indicated in describing the symptoms of the disease, and the characteristics of the patients suffering with it. Although organic disorders of all sorts are closely simulated, certain features will aid in distinguishing the hysterical condition. Primarily there is to be noticed the lack of proportion between the assigned cause and the symptoms produced; and a tendency for all symptoms to become worse when much attention is paid to them. There is also a grouping of symptoms which it is impossible to attribute to any known lesion or series of lesions. Yet in spite of this, the diagnosis from organic disease is often extremely difficult, and only careful study will suffice to exclude it. One of the chief difficulties is that the explanation of symptoms by the presence of hysteria does not occur to the physician encountering them so early in life. It is possible, too, to have a combination of hysteria with the evidences of an actual organic lesion, and this makes the diagnosis still more difficult.

Reviewing in more detail some of the diagnostic symptoms of the various manifestations, we find the major hysterical convulsive attack suggesting some form of epilepsy, but distinguished by the absence of biting of the tongue, of injuries done by falling, of real unconsciousness, of relaxation of the sphincters, and of terminal sleep; while the movements of the body are suggestive more of purpose, and the patient may later recall and speak of what took place. As regards other motor symptoms, hysterical contractures are marked by the greater rigidity on passive movement, as compared with those of organic nature. Hysterical paralytic conditions and contractures may disappear completely during sleep or under anesthesia. There is also no atrophy of moment or absence of the tendon-reflexes, such as should accompany some of the paralytic conditions of organic origin; and, as previously stated, there is often a distribution and an association with other symptoms which it is impossible to explain except on a hysterical basis. Hysterical chorea has often more violent and systematic movements than true chorea exhibits, and there is a tenseness of the limbs rather than the hypotonia of the latter disease.

Sensory symptoms exhibit similar suggestive characteristics. It is an important observation that through the diverting of the mind of the child, or during sleep, tender regions may entirely cease to be tender. Areas of hyperesthesia, too, do not show the distribution which organic lesions would necessarily produce. Hysterical involvement of the joints comes on more suddenly than one of an organic nature, often with more sensitiveness, with an absence of evidences of inflammation, and a greater variation in the symptoms. In hysterical headache no cause for the condition can be found, and the attack is liable to develop at a time when it may be of service to the child, or when suggestion is given by too much attention; and it disappears when something happens to entertain the patient or to divert the attention in other ways. The aphasia of hysteria is rather a dumbness. It comes on suddenly and there is no effort whatever to speak; such as would occur in many cases of true aphasia. In some instances, too, the patient can sing, whisper, or cough, although he cannot speak in the ordinary manner. Severe barking cough, or attacks of dyspnea occurring without sufficient discoverable cause, may frequently be determined to be hysterical from auto-suggestion, if the child at an earlier period has suffered from such symptoms depending upon actual organic cause. The repeated vomiting of hysteria is in many cases only partial, and not followed by the emaciation and loss of strength which one would naturally expect. Also suggestive are the ease with which the vomiting occurs, and the frequent apparently direct intention of the child. Hysterical anorexia may be traced to the earlier administration of some food very distasteful to the patient, or may be found to have been started by some actual dyspeptic disturbance and kept going by suggestion, often through the great urgency of the parents that the child should eat. Meteorism of hysterical nature disappears under anesthesia.

Treatment.—**Prophylactic treatment** is of the utmost moment. In the case of a child with nervous inheritance, or itself showing nervous manifestations, the greatest care must be taken that it lead a healthful life, free from all nervous excitement and from everything which calls attention to itself, especially with regard to ailments. A life in the open and in the country, with abundant exercise, is to be preferred. All possible reflex causes of nervousness should be removed by appropriate treatment; as well as all conditions which produce debility or lack of tone in the patient. The hours for sleep should be long, and the time for retiring early. Self-control must be taught; and all symptoms made light of. Undue mental work must be avoided and, indeed, the education must be made to occupy an entirely secondary place. Most important is it to remove the child from the care of a nervous, over-anxious mother. Nothing will sooner bring on hysterical symptoms in one predisposed than a constant noticeable anxious supervision of all the child's acts, with orders that this or that must not be done lest harm or illness follow.

All that has been said applies, of course, with even greater force to children who have at some previous time exhibited symptoms of hysteria; and applies, too, to the **direct treatment** of the disease already developed. Of especial importance here is the removal from the parents' care or from association with any nervous individuals. A judiciously selected nurse should be found who will be kind, and who will win the affection of the patient, but who will show no sympathy whatever. In fact, one of the best methods of treatment is to take no notice at all of the symptoms; or, at the most, to suggest on occasion that the patient is certainly better. The principle of suggestive treatment is of the greatest value in hysteria. Sometimes it is well to do nothing to entertain the child; sometimes better to fill its mind with matters which will divert it from a consideration of itself. A very careful study of the individual case is necessary, and no general rules can be formulated. Changes from time to time in the surroundings and in the things done for the patient are an important portion of the mental cure. Hydrotherapy, electrical treatment, massage, may follow one another; and do good partly by the improvement in tone, largely by the impression made. In some cases the rest-treatment is of service.

In certain selected cases, either the element of fear of pain or of other disagreeable sensation may be invoked, or the forcefulness of command can be employed in order to benefit through the element of sudden surprise. In the former the discomfort may be obtained by the use of the cold douche, counterirritation of the spine, application of a strong faradic current, or the like. The latter form of treatment consists in the sudden, sharp insistance by the physician that a paralyzed child get up at once; or the command to speak given to a case of dumbness, at the same time that a painful stimulant, such as an electric current, is applied over the larynx. This latter method is astonishingly successful when successful at all; but the danger of failure is great, and then the malady is harder to relieve. The method of ignoring the symptoms is more certain, although more tedious.

STIGMATA OF DEGENERATION

These consist of bodily or mental characteristics which are prone to be present in children with diminished power of resistance in the nervous system; nervous children; and those with neurasthenia, and especially with mental deficiency. They are not, however, necessarily present, nor when observed are they by any means positive indications of a nervous or mental weakness. They can be looked upon merely as corroborative signs. They are for the most part organic in nature, but are referred to here because at times associated with functional or psychic disturbances.

The *head* may be brachycephalic; dolichocephalic; macrocephalic; ydrocephalic; microcephalic; asymmetrical, or deformed in other ways.

The face may be asymmetrical; the lower jaw very prominent or markedly receding; the forehead abnormally narrow; the arch of the palate unusually high. Bifid uvula, harelip, and cleft-palate are to be included also. Among other deformities connected with the head are enlargement of the tongue; malposition or imperfect development of the teeth; deformity of the nasal bones. The eyes may show irregular coloring of the iris; congenital coloboma; albinism; opacities; congenital cataract; strabismus; irregular form or position of the pupils; oblique inclination or narrowness of the palpebral fissure; pigmentary retinitis. The ears may exhibit irregularity in shape; adhesion of the lobule; crumpling of the helix, or be unusually large or prominent.

In the region of the *trunk* are to be noted spina bifida; hernia; polymastia; deformities of the breasts. In the *genital organs* are epispadias; hypospadias; rudimentary penis; cryptorchidism; early development of genital hair; atresia of the vagina; and double vagina or uterus. The *limbs* may show such lesions as webbing or alteration in the number or formation of the fingers or toes; very long or very short arms, and the like.

The *development* as a whole may be altered; the child seeming prematurely old or having retarded puberty; or there may be evidences of feminism; masculinism; infantilism; gigantism; albinism; etc.

Functions, too, may show degenerative signs; to be mentioned here being delay in learning to sit or walk; defective speech; congenital deafmutism; stammering; enuresis; poor circulation of the blood; abnormalities of appetite, and diminished resistance to disease.

Among *psychical stigmata* are, in milder cases, the manifestations of nervousness, debility, and excitability mentioned in discussing nervous children; and in the severer cases the evidences of mental deficiency; moral insanity; sexual perversity; precocity in a limited direction; fixed ideas; phobias; truancy, etc.

MENTAL PRECOCITY

This condition in itself is in no way a disease; yet in many instances it is attended by characteristics which are distinctly pathological; and it may, therefore, receive brief mention here. A child who is precocious within normal limits shows merely an unusual and often decidedly rapid development of his intellectual powers as compared with his age. This may take place in all directions equally, or only, perhaps, along one or two lines; the mental condition being average in other respects. Precocious children may be born so, and advance without effort on the part of the parents, or even in spite of restraint. In many other instances there is at first little more than the normal degree of intellectual power; but over-prompt beginning of teaching of a forcing character, or association only with those older than themselves, develops in them an early acquisition of knowledge, and a precocity which is more apparent than real. Precocity of this sort is to be guarded against, as the premature stimulation of the intellect is liable to result in the development of some of the various characteristics of the nervous child. True precocity in all directions needs no checking except so far as is necessary to keep the bodily health in good condition. In the case of children precocious in certain directions only, great care should be taken that in the training along these lines others are not neglected. It is often better to make an effort to retard development on the precocious side.

Not infrequently a child possessing a wonderful talent of some one kind may be backward or even imbecile in every other characteristic. There are many interesting instances of this on record. Children of this sort need especial efforts to educate them along normal and varied lines.

PSYCHASTHENIA

Etiology.—This is a disorder allied on the one hand to endogenous nervousness (p. 269), but with predominance of psychic manifestations; while on the other hand, in its severer forms, it approaches closely to insanity. It is also in some instances related to those of the tics in which the movements are but a motor expression of imperative ideas. It is an inborn condition, in contra-distinction to neurasthenia; yet it may be dormant, and require some decided exciting cause to produce the symptoms. Among such causes are mental shock, sleeplessness, or exhaustion brought about in any way.

Symptoms.—The patients may be normal intellectually, possess special mental power, or verge on deficiency. They are nervous; imaginative; impressionable. The symptoms appear oftenest at, or soon after, puberty, but may develop in later childhood or even earlier. Among them are many of the psychic disturbances of the nervous child, but shown in an exaggerated degree. One of the most important is the feeling of doubt, and, closely associated with this, phobias of all sorts; and with these is combined an over-conscientiousness, which finally leads to obsessions and to imperative ideas. These result in hesitation in action and in reaching conclusions; lack of will-power and of concentration; restlessness; repetition of acts for fear they have not been properly performed; melancholic states from the consciousness of personal imperfection; hallucinations; fear of certain objects, places, and conditions; the fear of personal contamination, leading to frequently repeated washing of the hands and the like; impulses to spring into the water or out of a high window, etc. The disease may advance still further, and the obsessions and imperative ideas become so uncontrollable that criminal acts may be performed, such as theft or murder; or they may lead to self-multilation or suicidal attempts. Truancy from school or the running away from home is, in many instances, a symptom of psychasthenia; as is sexual perversion of various sorts. In fact some of these children appear to be moral degenerates. (See Moral Insanity, p. 300.)

Prognosis.—This is very uncertain, and unless treatment is instituted the disease is disposed to grow worse, although intermissions in its course may occur. Deterioration in health from any cause diminishes the resisting power and tends to increase the psychasthenic symptoms; and the surroundings of the patient have a very decided influence. Yet, under proper treatment, psychasthenia is often not incurable and the symptoms entirely disappear, even though the underlying tendency cannot be removed.

Diagnosis.—The diagnosis is to be made only from undoubted psychoses, such as insanity and imbecility. In psychasthenia the patient recognizes that the ideas, and the acts which follow, are wrong and not well founded, and consequently are to be concealed. He feels only an inability to control them; hence the title "imperative ideas." In a true insanity, on the other hand, the subject is unconscious of anything wrong in his concepts, logical processes, or deeds.

Treatment.—Measures are to be sought which improve the general health and quiet nervous excitability. All mental over-strain is to be avoided, and yet the child's mind must be kept filled by healthy ideas and occupations. Of special importance is the removal of the patient from unsuitable surroundings. Without this but little can be accomplished. The confidence of the child is without fail to be obtained, and then every effort put forth to increase the will-power. The attempt must be made to discover the nature and origin of the obsession which possesses the child, to remove the cause which occasions this, and to produce a conviction of the unreasonableness of the doubt or fear. Gymnastic exercises are useful both in improving the general condition, and in awakening the concentration upon healthy thoughts. In spite of lack of prompt results, treatment must be persevered with.

INSANITY

An exact definition of insanity seems almost impossible; and the various classifications made of its forms differ greatly one from another. The diagnostic distinction between idiocy and imbecility on the one hand and insanity on the other is also not always sharply made by psychologists. Perhaps a convenient construction is that insanity is the disease of a mind previously sound, coming on after mental power had been fairly well attained; while idiocy is a mental feebleness, depending upon a disease or defect of the brain, congenital or acquired during its development, which has arrested the growth of the mental powers (Peterson).[1] The symptoms of insanity are sufficiently characteristic in most cases. The confusion exists chiefly in regard to the form denominated dementia, which resembles imbecility in its symptoms, but differs in that it is a mental enfeeblement acquired later in life in a brain previously normal. Thus a mental failure following epilepsy in adults is spoken of as epileptic dementia; while, according to some psychologists, in children it is called epileptic idiocy on account of the mind being still in process of development. The distinction possesses, however, a certain degree of artificiality, and the subject needs further clarification.

Etiology.—Insanity is a comparatively uncommon condition in childhood. Spitzka[2] estimated that only about 4 per cent. of 3244 adult cases of insanity under his observation had developed the condition during childhood, and quotes Hagen that annually 1 in 70,684 children became insane in the region studied by him. Cases in very early life have been reported, even of congenital origin; but it is only in later childhood that the frequency of its occurrence begins to be significant, although the disease still remains uncommon until puberty is reached. Rhein[3] collected 39 cases, including 7 of his own, and excluding infectious psychoses, 14 being under 10 years and 25 between 10 and 16 years. There is often a decided inherited tendency observed; the parents being neurotic, or suffering from some nervous affection. Insanity may be a result of some lesion in the brain, as in cases of trauma, meningitis, or syphilis; or may oftener depend upon causes producing no organic intracranial lesions, such as chorea, epilepsy, acute infectious fevers, shock, and undue mental work, especially if these occur about the time of puberty. Excessive masturbation is doubtless a factor in some instances, but its influence is much over-rated. More often it depends itself upon the diseased nervous state which finally terminates in insanity.

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[1] Church and Peterson; Nervous and Mental Diseases, 1911, 851.
[2] Keating's Cyclop. Dis. of Child., 1890, IV, 1039.
[3] Amer. Journ. of Insanity, 1915, LXXI, 471.

Symptoms.—One of the most frequent forms of insanity is an *acute delirious state*, reaching even the degree of *mania*. It is encountered oftenest in connection with the acute infectious fevers, especially typhoid. The symptoms may develop either while fever is still present or during convalescence. There is great excitability; screaming; constant talking; and sometimes hallucinations or delusions. I recall a boy of 4 years with typhoid fever, whose screaming continued almost uninterruptedly for days, until he became exhausted and aphonic. Most febrile insanities last a few days to a few weeks, but sometimes longer; or rarely pass into a state of dementia. Mania may infrequently occur independently of any acute febrile process, and is then liable to recur in repeated attacks of short duration. I have observed an acute mania of excessive violence in a fatal case of chorea. Epilepsy may sometimes exhibit a transitory mania in place of, or following, the ordinary seizure. In other cases epilepsy of long-continuance and with frequent seizures may result in *dementia*. This is, however, more frequent after puberty. Dementia sometimes depends upon the cerebral lesions of hereditary syphilis, and is then seen in early childhood. Resulting from other causes it is rare under puberty.

Melancholia, or a melancholic condition, is one of the most frequent forms of insanity in early life. It occurs in later childhood, especially after mental over-work. There is depression; weeping; moodiness; sometimes hallucinations; and in some instances self-mutilation, or attempts at or even actual suicide. Sometimes the melancholic state alternates with maniacal seizures; in others it is attended by a stuporous condition.

An interesting variety is that designated *moral insanity*, in which, however, with the absence of moral sense there is often, but not always, associated a degree of intellectual defect. Even those who at first show no evidence of the latter are liable eventually to develop it. None of the restraints of religion or propriety seem to possess any influence, and even that of fear has but little power. All the tendencies are vicious; sexual impulses may be dominant and perverted; cruelty is a prominent feature; and as the child grows older he is liable to commit some criminal act. Strictly speaking the majority of these cases should be described as *moral imbecility*, since the disease is a congenital one, and the moral nature has been arrested in its development. There are, however, instances in which the moral perversion is the result of accident or disease occurring in the case of those who have previously shown no abnormality in this respect, and to these the title of moral insanity is properly applied.

Many of the perverted tendencies and feelings seen in neurotic children and in psychasthenia, such as fixed or imperative ideas and phobias, are present in increased degree in insanity. They differ, however, in the latter condition in that the patient does not recognize their unreality and groundlessness; and this is true also of the hallucinations and delusions of different sorts often present, in which the patient fully believes. Many of the stigmata of degeneration described (p. 296) are prone to be found in insane persons.

Prognosis.—This varies according to the cause and the form of insanity exhibited. In general it is better than in adult life. In melancholia and hypochondriasis recovery generally takes place, as also in the acute delirious insanities and the insane state sometimes attendant upon hysteria or chorea. The dementia following syphilis or long-continued epilepsy offers an unfavorable prognosis. Forms of insanity with fixed delusions are unfavorable, and moral insanity equally or even more so. Always to be remembered is the liability to recur in cases apparently cured.

Treatment.—Merely can be urged in this connection the maintenance of the general health; the removal of the cause if discoverable; the separation of the patient from unfavorable surroundings; and the careful guarding against injury done by the patient to himself or others. The details of treatment vary with the form of the disease, and are more properly considered in works upon mental disorders.

IDIOCY

(Imbecility; Feeble-mindedness; Deficiency)

Definition and Classification.—As with insanity the classifications of these conditions are numerous and confusing. The distinction from insanity has already been defined (p. 299); idiocy being regarded as a mental feebleness due to organic cerebral defect or disease present at birth, or arising in the brain while the mental powers are in a developing state. The idiocy of cretinism constitutes an exception, the mental arrest in this case being a symptom of disease of the thyroid gland. This condition will be described separately.

Except for sentimental reasons, the terms idiocy, imbecility and feeble-mindedness can all be included under the title "idiocy." Yet this title, as well as that of "imbecility," is one almost of opprobrium among the laity, and with propriety avoided by the physician in speaking to parents. The three varieties mentioned constitute merely degrees of the same clinical condition, with all sorts of connecting grades between them; the typical "idiot" of common parlance being at the one end of the scale, with a mental condition little above or often inferior to that of the lower animals; the slightly feeble-minded or "deficient," called also the "moron," at the other end, but little separated from the normal individual. There is further a moral imbecility, usually combined with some degree of intellectual deficiency. This has already been discussed under Insanity (p. 300).

The term *backwardness* is often applied in a rather loose manner to children who at a certain age have not attained to the standard of mental development natural to this. Many of these children should properly be called feeble-minded; but in other instances the backwardness depends upon an attendant slowness of physical development, perhaps the result of debilitating diseases; or upon a lack of training, defective eyesight or hearing, the presence of adenoid growths, and the like. All such cases are capable of cure, and are not properly to be included among the mentally deficient. Allied to these are the cases of *idiocy by deprivation,* in which distinct mental defect of mild degree is the result of physical disorders which prevent the proper development of the mind. This is best illustrated in congenital deaf-mutism. Apart from the types already referred to, numerous other classifications have been proposed. Some rest largely on a clincial basis; some on an etiological; and others on a pathologico-anatomical one. A general division sometimes employed is that of Langdon Down[1] into the *congenital, developmental,* and *acquired* forms. The first includes cases in which the symptoms can be recognized not long after birth. The second comprises those developing in the process of growth. The third contains a large variety in which are such cases as those dependent upon trauma at or after birth; convulsions occurring in infancy; meningitis; hydrocephalus; infectious diseases; and epilepsy.

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[1] Tuke's Dict. of Psycholog. Med., 1892, 644.

Other classifications separate the forms chiefly on the basis of anatomical characteristics, or on that of a combination of these with etiological factors. Thus we have idiocy which is microcephalic; hydrocephalic; paralytic; epileptic; mongolian; traumatic; inflammatory; toxic, deprivative, etc. A few of the many varieties mentioned by writers will be briefly discussed later. Fuller descriptions will be found in works upon psychiatry.

Etiology.—The condition is a common one. Reuben[1] maintains that there are about 150,000 mentally deficient individuals in the United States, not including the insane and the epileptic; and Stowell[2] places the number even higher, assigning 1 feeble-minded to every 300 normal persons in the United States. *Inheritance* plays a very important rôle and shows itself in from 40 to 50 per cent. of the cases (Peterson).[3] The parents may themselves have been degenerates, or only neurotic, or the subjects of nervous disease of some sort. Alcoholism in the parents is a decided factor, as in tuberculosis; with syphilis to a less degree. In 204 cases of idiocy studied by Atwood[4] 14.7 per cent. gave a positive Wassermann reaction. However, the statistics in general upon the influence of syphilis vary much. Any condition which debilitates the parental health before the birth of the child exercises some etiological power. Consanguinity in itself does not possess the influence often assigned to it, unless both parents are neurotic, in which case the tendency to all nervous conditions in the offspring is increased. In the large majority of cases the symptoms appear at an early *age*. *Sex* is a factor also, male idiots preponderating in the proportion of about 2 : 1.

Trauma occurring at birth is a factor of great importance; in the paralytic cases somatic evidences of an intracranial lesion existing in combination with mental defect. Thus meningeal hemorrhage with its mental sequels results from prolonged labor, instrumental delivery, or long-continued asphyxia after birth. It is to be noted in this connection that the employment of the forceps is an element of far less importance than is prolonged labor. In fact, a sufficiently early use of these would probably prevent the development of many cases of cerebral paralysis with idiocy. Cases of *premature birth* are liable to be found mentally defective later; probably either from lesions of the weak-walled vessels occurring as a result of this; or on account of the imperfect development of the brain at the time of birth.

The cases of idiocy resulting from causes acting after birth are much fewer. Prominent among these are eclamptic convulsions, a steadily increasing mental degeneration sometimes dating from a convulsive attack. Among other post-natal causes are to be mentioned epilepsy, febrile diseases, trauma, mental shock, and mental over-work.

Pathological Anatomy.—Organic lesions of some kind are usually at the basis of idiocy, but these are of great variety, and none of them characteristic. They may be of a gross nature, or only discoverable by microscopical study. Among those of the latter sort, there may be the condition described as agenesis corticalis (Sachs)[5] (see p. 310), in which there is an arrest of development of the cortex with an absence or degeneration of the normal cortical cells and fibres. In another class there is sclerosis of some sort; or the lesions following encephalitis, meningitis, hemorrhage, thrombosis, embolism, and tumors. Often the brain is much altered in shape, or there are defects of large size; as in hydrocephalus, porencephalus, general atrophy, absence of a hemisphere, development of cysts, etc.

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[1] Arch. of Ped., 1912, XXIX, 596.

[2] Arch. of Ped., 1913, XXX, 203.

[3] Church and Peterson; Nervous and Mental Diseases, 1911, 860.

[4] Jour. Amer. Med. Assoc., 1910, II, 464.

[5] Journ. Nerv. and Mental Dis., 1887, XIV, 541; 1892, XIX, 663.

Symptoms.—Viewing these merely in a general way, there is first to be noted in all classes the predominant feature of a distinct enfeeblement of mind; an inability to fix the attention properly; and a consequent diminution in the power of receiving impressions and forming concepts or reaching conclusions. These symptoms appear early in well-marked cases. With them are frequently combined a lack of proper physical development and some of the stigmata of degeneration. Some alteration in the shape of the head or the expression of the face is nearly always present. An unusually high V-shaped arching of the vault of the palate has been claimed to be a suggestive somatic characteristic; but Channing's[1] observations in a study of 1000 idiots confirm the opinion of others that this anatomical deformity appears to possess no real significance. Superficial inspection is sufficient to render a diagnosis possible in nearly every instance. In the typical *idiot* of low grade the mental faculties are more or less completely impaired; there is inability to say more than a few words, if any, or to understand anything said. The infant cannot recognize objects, and takes no note of any put before him. Later in life he is less capable than the lower animals of guarding against ordinary dangers. Much after the normal age for doing this, he is still unable to sit up or even to hold the head erect; the saliva dribbles from the mouth; and the ability to walk comes very late or not at all. Blindness is present in a certain not large percentage. In the case of *imbeciles* the power of receiving impressions is greater, but that of fixing the attention is still very poor. The imbecile is slow in learning to walk, but attains the power completely. He may learn to talk fairly well, but only after much time has elapsed and after continued teaching. He understands to a degree, and can obey orders if so disposed; but the behavior is unreasonable and mischievous, and the child may be subject to attacks of maniacal excitement or rage. This somewhat vicious character, although common, is not observed in all imbeciles, some of whom are good-natured and industrious. Imbeciles are able to feed and dress themselves. They are commonly extremely restless and uncontrollable; and in the office of the physician or the class room they are in no way awed by the strange surroundings, but run about and handle everything, often destructively, in a manner which is very characteristic. Automatic or rhythmic movements are common. Some imbeciles exhibit remarkable mental power or talents in certain directions. (*Idiots savants*, see p. 306.) In others the intellectual defect is combined with a moral imbecility. The practice of masturbation is extremely frequent and often excessive.

These conditions of typical idiocy and of imbecility shade into each other, and there are, as already stated, all grades from these to mere *feeble-mindedness* or *deficiency* of various degrees. Children of this last class (*morons*) speak usually without difficulty, are slow in acquiring ideas, and never become able to reason quite normally. They can learn but little at school, and even in the mildest cases are always behind their classes. They may under favorable conditions later become self-supporting; but under ordinary circumstances are unable to cope with others in business matters. Very readily they drift into crime, the women often becoming prostitutes.

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[1] Journ. Mental Sci., 1897, Jan.

Varieties.—But a few of the many clinical varieties of idiocy which have been described may be briefly mentioned here in more detail. Some of them have been especially well discussed by J. Thomson,[1] from whom I have quoted freely.

1. Paralytic Idiocy.—This is the form dependent upon lesions producing infantile cerebral paralysis. The degree of mental defect is not always in proportion to the completeness and extent of the paralysis, but it is prone to be greatest in the diplegic cases. It dates usually from a meningeal hemorrhage occurring during birth; or less often from this or some other lesion developing at a later period. The evidences of paralysis present are of a spastic nature and with the distribution seen in cerebral palsy (p. 365). The degree of mental defect varies from decided idiocy to a slight deficiency. Porencephalic idiots exhibit spastic paralytic symptoms of a similar nature.

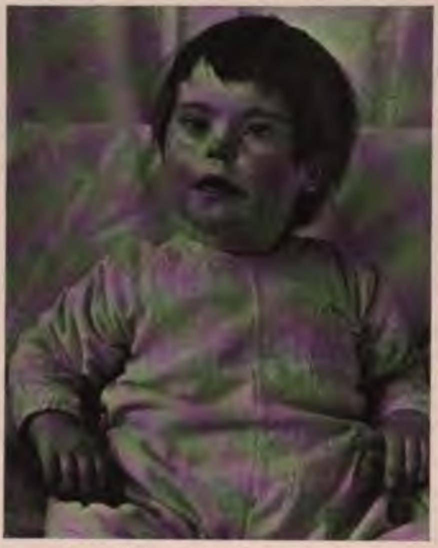

FIG. 328.—MONGOLIAN IDIOCY.
Patient in the Children's Hospital of Philadelphia.

2. Mongolian Idiocy.—In this well-recognized clinical variety, which constituted 20 per cent. of 750 collected cases of mental deficiency published by Reuben,[2] there are some peculiar somatic conditions observable. The skull is small and brachycephalic; the occiput flattened; the nose flat and broad; the mouth usually small and open, and adenoid growths are common. The tongue may be enlarged, and it is prone to protrude (Fig. 329) even although of normal size, and later is fissured. Sucking of the tongue is a symptom present in nearly all cases. The palpebral fissures are narrowed and slope upward outwardly, with an

[1] Scottish Med. and Surg. Journ., 1898, March, 203.
[2] *Loc. cit.*

unusual development of the inner epicanthic fold, suggesting the Mongolian type of face (Fig. 328). Growth in these children is very slow, and they never become of normal size. Those who live to puberty are prone to become very fat. The teeth are late in appearing, small, and decay early; and the learning to walk and talk is much deferred. The hands are often very characteristically altered, being broad and square, with the fourth finger and the thumb abnormally short, the former being in-curved toward the third finger. The feet, too, are broad and flat. The limbs are rather short. There is a general flaccidity of the muscles and ligaments which often allows of unusual passive movement. The general strength is below normal, and the resistance to disease is slight. There is a rather marked tendency to congenital heart disease as a complication. The children are usually good-natured and lively. The cause of Mongolian idiocy is obscure and the lesions not characteristic. It is to be noted, however, that a large proportion are born of mothers near the close of the child-bearing period and who have had a number of pregnancies.

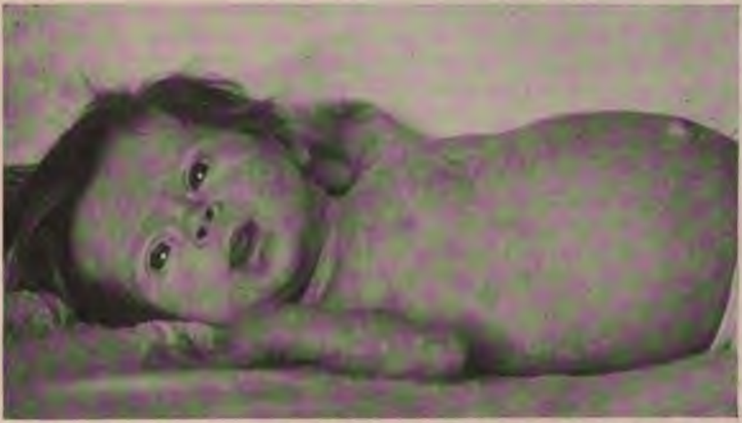

FIG. 329.—MONGOLIAN IDIOCY, SHOWING PROTRUSION OF TONGUE.
Patient in the Children's Hospital of Philadelphia.

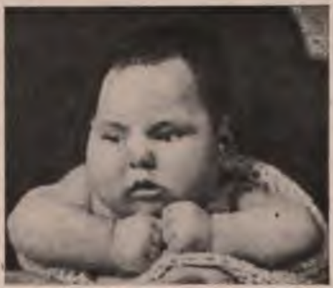

FIG. 330.—MICROCEPHALIC IDIOCY
Child of 4¾ months. (*Thomson, Clinical Examination of Sick Children*, 1908, *2d Ed.*, 55.)

3. Microcephalic Idiocy (*Aztec Type*).—(See also p. 309.) In these patients (10 per cent. of Reuben's 750 cases) the most striking anatomical feature is the remarkable smallness of the cranium. The forehead is very narrow, the vertex pointed, and the occiput flat (Fig. 330). The bones of the skull are often prematurely ossified, and the sutures and fontanelles close early. In other respects the infants are usually well-formed and well-nourished. The mental power in these children is very defective, and they belong in most cases to the class of typical idiots in which little improvement can be expected. A marked degree of muscular rigidity is often present, the arms being held firmly against the sides;

the elbows, hands and fingers flexed; and the legs stiff and adducted. Less severe cases are without these spastic symptoms, and may be taught to walk and even to talk a little. In microcephalus, as is now generally recognized, it is not the case that the brain does not grow because the cranium is small, but the etiological relationship is the reverse of this. Consequently craniotomy can be of no benefit. I have seen it performed in a number of instances without improvement.

4. **Hydrocephalic Idiocy.**—It is of frequent observation that children with hydrocephalus may be very bright; and even severe cases may have a remarkable preservation of intellectual power as compared with the amount of brain substance present. Sooner or later, however, with the increase of pressure of the fluid, there is exhibited mental impairment of varying degree.

5. **Epileptic Idiocy.**—Here the idiocy is often a slow development after the long continuance of epilepsy, and usually does not appear before puberty. A gradual mental deterioration takes place, with the symptoms of dementia, which in older persons would properly be classified as insanity. The condition may be consecutive to epileptic mental disturbances of a more active form.

6. **Amaurotic Family Idiocy.**—Much interest has been experienced in the condition to which this name was given by Sachs,[1] although the alterations in the macula had been previously observed by Tay.[2] Numerous cases have since been reported. It is seen usually in Hebrew infants, and often several in a family are the subjects of it. The ordinary evidences of idiocy become apparent generally in the latter half of the 1st year, up to which time the child had seemed entirely normal. Now it grows weak; apathetic; lies quietly in bed and loses the muscular power which it had attained; and the condition of stupidity gradually increases. The point of interest lies in the condition of the eyes. Study of these shows that the child cannot see, and ophthalmological examination reveals characteristic changes in the disc and the macula lutea in both eyes. The normal appearance of the latter is replaced by a reddish spot surrounded by a bluish-white halo, and there is more or less atrophy of the optic nerve. Progressive deterioration of the child's strength and general condition follow. The muscular flaccidity at first seen is replaced by a spastic state with increase of the patellar reflexes. Convulsions may occur and death will generally take place within a year.

The pathological lesions in the brain consist in degenerative changes principally in the cells of the cortex; but the grey matter of the entire cerebrospinal axis may be involved.

7. **Idiots Savants.**—This includes a class of idiots in which there are special bodily or mental powers in certain limited directions. Many celebrated cases are on record, in which these powers have far surpassed those of the average individual. Thus there may be remarkable aptitude for music, painting, mathematical calculations, mimicry, games, or other accomplishments; yet the individual is not above a condition of feeble-mindedness, or even of imbecility, in all other directions.

In addition to the classes of idiocy described there are a large number (over 50 per cent., Reuben) which do not belong to any definite type.

Prognosis.—The chance of ultimate complete recovery in those whose minds are actually defective can hardly be said to exist; but the very great possibility of errors in diagnosis in infancy is not to be for-

[1] New York Med. Jour., 1896, LXIII, 697.

[2] Transac. Ophthalm. Soc. of the United Kingdom, 1881, I, 55.

gotten. It is also true that under proper care and training many cases are susceptible of great improvement; and that a long time must pass before it can be stated that this will not occur. The anxiety of the parents is naturally great, and an adverse opinion is to them often worse than the assurance of the child's early death. From all these points of view, then, the prognosis should be made guardedly. That as to improvement depends upon the nature of the cause, the variety of idiocy and its degree, and the care taken in treatment. The low-grade idiot can improve but little, but may be made less offensive and the imbecile less destructive. The microcephalic idiot rarely lives to adult life and usually does not survive infancy; yet instances are on record in which life has continued for many years. The amaurotic family form gives an entirely unfavorable prognosis, and is usually fatal at an early period. Mongolian imbeciles are liable to fatal attacks of various illnesses, and only few live beyond the period of puberty. The improvement which takes place in them is very decided for a time, but then ceases and the patients, although learning to talk to a limited extent, never pass the stage of low-grade imbeciles. Imbeciles of any class can be taught to care for personal cleanliness, to talk, and often to learn some occupation. Idiots savants are liable early to suffer from increasing mental degeneration. Epileptic and paralytic idiots of low grade, and any with gross anatomical cerebral lesions, are seldom capable of much improvement. In general, cases of severe mental defect are usually short-lived, dying in childhood or adolescence. Subjects of mere feeble-mindedness or backwardness are capable of remarkable improvement to an extent which cannot at first be predicated. To accomplish this, however, the best of training is required, preferably in an institution.

The prognosis is also influenced by the time at which the idiocy developed and the nature of the symptoms. The earlier in life mental defect becomes apparent, the worse the prognosis. Hence the congenital cases, other things being equal, are more unfavorable than others. Great restlessness renders the prognosis for intellectual improvement more unfavorable, through the difficulty of claiming the child's attention for purposes of instruction.

Diagnosis.—The diagnosis is easy in all well-marked cases past the age of early infancy, and there are often features which clearly proclaim the type. In all of them there is something wrong about the general appearance which is apparent to any one. Often there is a malformation of the head which is a sufficient indication, and the expression of lack of intelligence is characteristic. In the milder cases, however, the diagnosis often presents difficulty. To form it correctly it is necessary to know the ages at which the normal child should begin, respectively, to notice, sit, stand, walk, talk, and to exhibit other signs of intelligence. This has already been discussed under the Physiology of Infants (Vol. I, p. 65). Later in childhood the application of the Binet-Simon and other tests is of great service for determining the degree of backwardness as compared with other children of equal age. (See Dana,[1] Knox,[2] and Goddard.)[3] Before reaching a diagnosis of mental deficiency, however, it is important to entertain the possibility of some other condition being present. Thus blindness or deafness may be the cause of lack of observation and attention, and a careful study of the hearing or sight should be made, including here an examination of the nasopharynx for adenoid growths. Slowness in bodily or mental development may be but a temporary matter, the result of malnutrition of some sort. Some children, otherwise normal, learn to speak late, although understanding well; this depending often upon a lack of desire on the part of the child to take the trouble to express by language what he can do more easily in some other way (p. 280). Great slowness in learning to sit or walk may be caused by severe rickets. The exculsion of all of these possibilities is necessary before a diagnosis of mental defect is justifiable. In general, a child who has not learned to talk at all by the age of 2½ years probably is suffering from some actual mental defect, particularly if the understanding of words is apparently not normal. The presence of any of the stigmata of degeneration is a corroborative diagnostic symptom of idiocy, and the existence of exaggerated knee-jerks or of a very slight rigidity or paresis may confirm the diagnosis of the paralytic form. The Mongolian type possesses features which sometimes makes it readily confounded with cretinism. The fingers, however, are not so square at the tips; some of them are shortened in the manner described; there are not the deposits of fat about the clavicles which occur in the cretins; the skin is softer; and the neck is thinner.

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[1] Med. Rec., 1913, LXXXIII, 1.

[2] New York Med. Journ., 1914, XCIX, 527.

[3] The Training School, 1910, Jan.

Treatment.—For low-grade idiots little more can be done than to attend to the condition of the general health, to care for cleanliness, and to guard against injury. Especially where there are other children in the family it is much better to remove the subject from the house. For those less seriously affected, an astonishing amount of good can often be attained by careful treatment. This is, however, a matter requiring such constant attention and training that special teaching and skilled instruction are needed to obtain the best results. The underlying principles consist in the effort to increase the interest and power of attention; to awaken the association of objects with the names for them; and to teach the employment of words, and the use and control of the muscles. These efforts cannot be commenced too early, although during the first 2 years of life they must not be employed to a degree sufficient to fatigue or worry the patient. For the details of treatment reference must be made to works upon psychiatry.

CHAPTER IV

DISEASES OF THE BRAIN AND MENINGES

Here are included, for the most part, those affections in which there is organic disease chiefly marked in or limited to the brain and its membranes. Certain affections which involve the spinal cord as well are preferably considered later. The classification which follows is largely a matter of convenience from a clinical standpoint.

MALFORMATIONS

Anencephalus.—Infants born with this condition present an absence of the cerebral hemispheres, an atrophic condition of the cerebellum, and an absence of the vault of the cranium (Fig. 331). It is often combined with other malformations especially of the spinal cord. There is a familial tendency to it sometimes seen. The malformation is incompatible with life for more than a few days.

Cyclopia.—The cyclops monster has the vault of the cranium present, but the hemispheres imperfectly formed, united anteriorly, and perhaps cystic. There is in typical cases but a single eye in a median position. Other malformations may be present, especially those of the face, the nose being only rudimentary. Well-marked cases die shortly after birth; but in those in which the malformation is only partially developed life may continue through childhood.

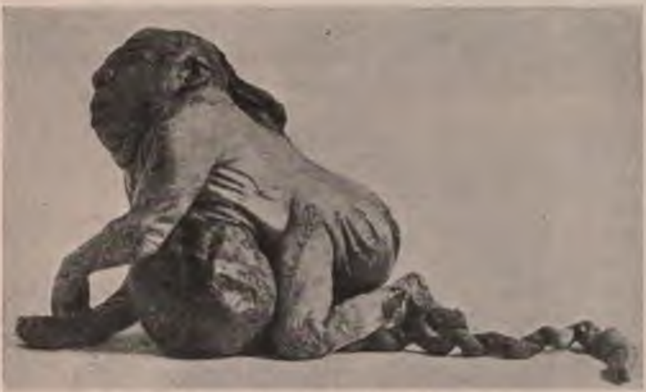

FIG. 331.—ANENCEPHALUS.

A 5-months fetus, with large ventral hernia, and imperfect development of the spina column. (*Merrill, Arch. of Pediat.*, 1906, *XXIII*, 390.)

Microcephalus.—This malformation has already been referred to as characterizing certain forms of idiocy. (See p. 305 and Fig. 330.) The head is unusually small, measuring at the end of the 1st year sometimes less than the normal birth-circumference. (See Vol. I, p. 32.) It is also altered in shape in various ways. Oftenest it is brachycephalic, or it may be dome shaped, or the forehead unusually sloping or narrow. The fontanelles are closed and the sutures united. The brain is correspondingly smaller than normal, and imperfectly formed in portions, or exhibits signs of degeneration. The primary condition is the smallness of the brain, and that of the cranium is secondary to this. In some instances the circumference of the head may be normal, but the brain exhibits the characteristics of microcephalus, the remainder of the cranial cavity being filled with fluid. The symptoms and prognosis have been discussed under Idiocy. (See p. 301.)

Porencephalus.—Here there is a distinct absence of some of the cerebral tissue, producing a depression on the surface of the brain, which sometimes extends into the lateral ventricle, the surrounding regions being normal. The interior of the affected region may be cyst-like and contain serous fluid, or no cavity may be present. The convolutions of the adjacent cerebral substance often seem to radiate from the depression. A portion of, or even a whole, hemisphere may be involved in the depression (Fig. 332), or the porencephalus may exist in both hemispheres. The condition may be congenital or acquired. In the acquired cases it depends upon a degenerative process following inflammation, thrombosis, embolism, or hemorrhage. In the cases developing before birth it is uncertain whether these agencies may also be the active factors, or whether the cavity-formation always results from a failure in development. In the congenital cases the lesions are seen oftenest in the anterior or middle portion of the brain and may be on both sides; or may be unilateral and associated with unilateral deformities of the skull. In the acquired cases, also, the lesions may involve one or both sides. The **symptoms** of porencephalus vary with the position of the lesion. More or less mental deficiency is generally present in cases of large cerebral defect, combined with the symptoms of spastic cerebral palsy, hemiplegic or diplegic in type. (See p. 366.) Disturbances of sleep and epileptic convulsions are sometimes observed. The duration of life is variable, depending upon the size and position of the lesion. In large defects death usually takes place in infancy, and the majority of all patients die before adult life is attained.

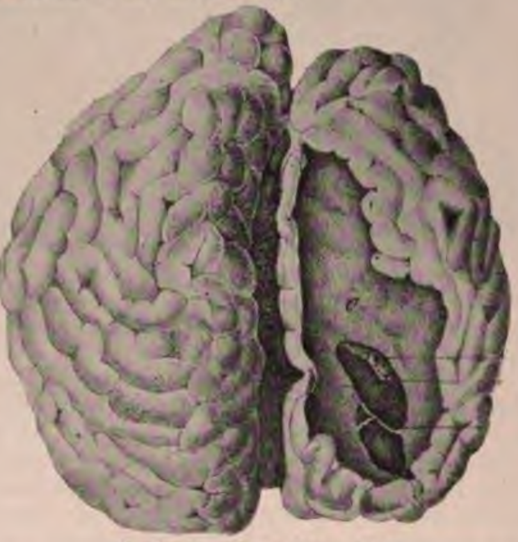

FIG. 332.—PORENCEPHALUS.

Cavity occupying a large portion of the right hemisphere. (*Ferraro, Riv. Internaz. di med. e. chirurg.*, 1886, *III*, *Pl. III*, *after p.* 506.)

Hydrocephalus.—Although some cases are acquired, the greater number are congenital as far as the cause is concerned, although there is, as a rule, no evidence of the condition at birth. The subject is considered later among other diseases of the brain (p. 356).

Macrocephalus.—This is a very rare congenital affection in which the size of the brain is increased. The skull may be enlarged; the convolutions anemic and flattened; the ventricles compressed or obliterated. The symptoms are chiefly those of hydrocephalus.

Nuclear Aplasia.—This lesion consists in a defective development of the cranial nerve-nuclei. It effects oftenest those governing the ocular movements, but in some cases the expression of the face and the movements of the tongue are involved. (See Atrophy and Sclerosis of the Brain, p. 361.)

Agenesis Corticalis.—There is here a congenital failure of development of the cells of the cortex, especially the pyramidal cells (Sachs).[1] It involves all parts of the cortex of both hemispheres. It is one of the causes of infantile cerebral paralysis, as also of idiocy.

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[1] Jour. Nerv. and Mental Dis., 1887, XIV, 541.

ENCEPHALOCELE; HYDRENCEPHALOCELE; MENINGOCELE

Etiology.—The cause of these comparatively unusual malformations is not clearly understood. In some way a defect in development has occurred during fetal life. It is possible that hydrencephalocele is the remains of a fetal hydrocephalus, as claimed by Spring.[1]

Symptoms.—Certain features in common characterize these three conditions. In all there is an opening in the skull and dura mater, usually of small size, through which a portion of the brain or of its membranes protrudes. The opening is oftenest between two of the bones rather than in the substance of one. There results a tense, smooth tumor, globular or pyriform in shape, generally with a narrow pedicle, and varying from perhaps an inch (0.39 cm.) in diameter to a size equalling that of the infant's head. This may be in different parts of the skull, but most often it is in the occipital region in or near the median line, and sometimes almost as far posteriorly as the foramen magnum. The frontonasal region, slightly to one side of the median line, is another favorite position. The malformation here may involve the root of the nose. Sometimes it projects into the pharynx or the mouth. When external it is covered by skin, which is sometimes of normal texture; often thin, shining, and without hair, and sometimes exhibiting cicatricial markings.

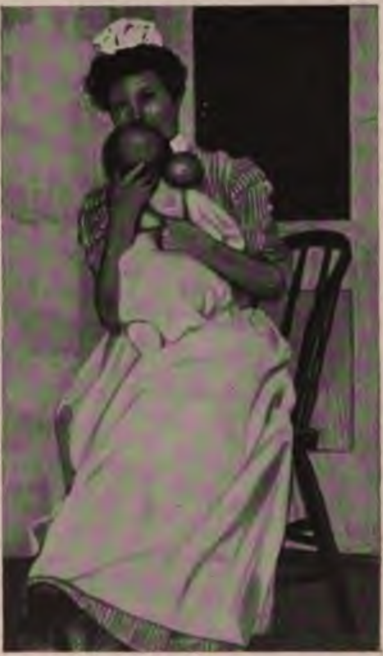

FIG. 333.—MENINGOCELE.

Female infant, aged 4 months. Tumor present at birth, and increased in size. Operation. Recovery. (*Wellington, Arch. of Pediat.*, 1907, *XXIV*, 115.)

In *encephalocele* (*hernia cerebri*), an uncommon variety, the tumor contains brain-substance and membranes, is usuallly small, without pedicle, opaque, pulsates but does not fluctuate, and is generally reduceable. In *meningocele*, also an uncommon form, the opening in the bone is usually smaller, the tumor contains only the meninges distended by cerebrospinal fluid, and communicates with the arachnoid cavity with occasional exceptions (Fig. 333). It is fluctuating, pediculated, does not pulsate, becomes more tense on crying, and is translucent and reduceable. In *hydrencephalocele* (*encephalocystocele*), the most common form, the tumor contains brain-substance with cerebrospinal fluid within it, which communicates with a ventricle. It is generally of larger size and provided with a pedicle, is only partially translucent, fluctuates, is partially reduceable, and often pulsates. Taking all forms together the majority occupy the occipital region, with the frontal region the next most common seat. In Houel's series[2] of 93 cases, 68 were occipital,

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[1] Thèse de Paris, 1853, 86.

[2] Arch. gén. de méd., 1859, XIV, 409; 569.

16 frontonasal, and 9 basal; and in Reali's[1] 140 cases there were 86 occipital, 33 frontal, 12 sagittal, 8 lateral, and 1 basal. Deformities of other sorts are often associated with any of the forms; among them spina bifida, cleft-palate, harelip and club-foot.

The general symptoms depend upon the size, position and nature of the tumor. Meningocele is without nervous manifestations, except that the effort at reduction may produce evidences of intracranial pressure. Encephalocele may also be free from symptoms except those of pressure produced in this or in other ways. Hydrencephalocele, on the other hand, is liable to be attended by hydrocephalus, optic atrophy, idiocy, nystagmus and strabismus. This is especially true of the tumor when in the occipital region. In addition to these symptoms may be found local ones, such as interference with respiration when the malformation involves the interior of the nose or when it is situated in the pharynx or mouth.

Prognosis.—This is on the whole unfavorable. In meningocele the tumor, although small at first, grows larger and there is always danger of rupture with subsequent secondary infection. Yet a chance exists in favorable cases that the communication with the arachnoid cavity may become closed. Encephalocele offers the most favorable prognosis, since it increases but little in size. In hydrencephalocele the tumor is large and liable to secondary infection or to rupture, and the prognosis is entirely unfavorable. The duration of life in these malformations is usually short, and many cases die within a few weeks. If of small size and properly protected, life may continue much longer.

Treatment.—In the cases of meningocele a small quantity of the fluid may be removed by aspiration if rupture is threatening, and an application of flexible collodion made to favor contraction; or a metallic protective dressing may be used to prevent compression or accidental rupture; or a plastic operation may be done to close the opening. Encephalocele should merely receive protection. Hydrencephalocele may be subjected to plastic operation if rupture is threatening, on the ground that it offers the only hope; but the results are usually unfavorable.

Spurious Meningocele.—There exists in addition to the forms mentioned a spurious meningocele, the result of trauma producing fracture of the skull and rupture of the dura mater in young children, union of the opposing fractured edges having failed to take place. Operative treatment may be of avail. A study has been made by Sailer[2] based upon 37 collected cases.

DISEASES OF THE MENINGES OF THE BRAIN

EPIDURAL HEMORRHAGE

This is a rare condition, almost always the result of trauma which has ruptured branches of the meningeal arteries. Compression of the head during labor is the most common traumatic agent; but fracture of the skull after birth is likewise an etiological factor. The blood is effused between the dura and the skull, producing an internal cephalhematoma, analogous to external cephalhematoma (Vol. I, p. 269), with which it, indeed, may sometimes be combined. The symptoms are those of gradually increasing cerebral compression, beginning in from a few hours up to a

[1] Behandlung angeb. Schädel-oder Rückgratsbrüche, 1874. Ref., Lindfors, Volkmann's Sammlung klin. Vorträge, 1897–1900, Gynäkol., No. 280.

[2] Univ. Med. Magaz., 1900, XIII, 515.

day after the injury, and characterized by slowness of pulse and respiration, with coma, convulsions, and paralysis. The diagnosis of the condition from other forms of meningeal hemorrhage is difficult. The symptoms are generally slower in their onset than in the case of subdural or subarachnoid hemorrhage. Death takes place usually within 24 hours. The only treatment possible is of a surgical nature, and this may relieve if undertaken promptly; yet many cases will die in spite of it.

PACHYMENINGITIS EXTERNA

In any of its forms external pachymeningitis is of very uncommon occurrence in children. A *chronic form* has been reported as a tuberculous or syphilitic involvement of the dura secondary to the appearance of these diseases in the skull; and is also sometimes represented by fibrous adhesions to the skull, the accompaniment of a chronic leptomeningitis. *Purulent external pachymeningitis* is nearly always secondary to an acute purulent process elsewhere, especially one of the middle ear which has involved the mastoid cells. Sometimes the original disease is within the bones of the skull, as in the frontal or other nasal sinuses. The epidural abscess which develops may remain of small size, and the inflammation localized; or may become extensive, and involve secondarily the interior surface of the dura (*purulent internal pachymeningitis*). In the latter event the pia-arachnoid is almost always quickly involved as well.

The **symptoms** are sometimes slight and entirely concealed by those of suppuration of the middle ear. In other cases they are severe, consisting of headache and evidences of cerebral compression, such as optic neuritis, vomiting, slowness and irregularity of pulse and respiration, somnolence, or coma; none of which distinguish it from cerebral abscess. There are, however, less often localizing symptoms than in the latter disease. Pachymeningitis is also to be distinguished from localized purulent leptomeningitis. Operative exploration is the best means of differentiating between the two, as well as the only treatment; and this should be undertaken promptly.

PACHYMENINGITIS INTERNA HEMORRHAGICA; SUBDURAL HEMORRHAGE

Etiology.—The disease is uncommon in early life. Among the most frequent causes are traumatism, as at birth; syphilis; severe infectious diseases; rickets; advanced cachectic states; hemorrhagic disorders; and affections of the heart and lungs. In 57 cases collected by Herter[1] 43 were less than 1 year old.

Pathogenesis and Pathological Anatomy.—The two conditions designated in the title are closely allied, and much discussion has arisen over their relationship. The older theories described the primary lesion as a subdural hemorrhage, upon which there was afterward engrafted an inflammation. A later theory, dating from Virchow[2] and now more generally accepted, designates the pachymeningitis as primary, with which may be secondarily associated more or less subdural hemorrhage. There would appear to be cases of primary subdural hemorrhage without inflammation, as there certainly are others of pachymeningitis with very little effusion of blood; and there are mixed forms. Very probably in many instances which appear to be hemorrhage the early traces of inflammation have been obliterated. In the recent inflammatory cases there is found a very delicate, highly vascular and easily removable soft membrane deposited upon the inner surface of the dura mater. It is situated oftenest over the convexity of the hemispheres, on one or both sides. A frequent position also is at the base. Hemorrhages found on the affected dura are usually punctate and scattered, with some of larger size; but when hemorrhage constitutes the predominating lesion a large effusion of blood covers portions of one of both hemispheres (*hematoma of the dura mater*), and little or no trace of false membrane may be discoverable. After a time changes take place in the lesion; the effused blood clotting and then partially organizing, and later being absorbed and leaving cysts; or the false membrane thickening into several layers and becoming more adherent to the dura or even binding it to the pia.

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[1] Amer. Journ. Med. Sci., 1898, CXVI, 202.

[2] Verhandl. phys.-med. Gesellsch. in Würzburg, 1857, VII, 134.

Symptoms.—These are uncharacteristic and often unrecognized, especially in marantic infants and those with severe diseases of other nature; the diagnosis under these circumstances being made only at autopsy. Any present depend generally upon the hemorrhage rather than upon the inflammatory process, and consist of vomiting, headache, convulsions, unconsciousness, and later indications of compression; among the last being bulging of the fontanelle, slowness and irregularity of the pulse and respiration, choked disc, stupor, convulsions, and coma. Fever may be among the early or later symptoms, or may be absent. In cases of long duration with cystic formation the head becomes decidedly enlarged. Evidences of paralysis and rigidity may appear, the locations of these depending upon the seat of the lesion.

Course and Prognosis.—Death may take place within a week, or the condition may become of a more chronic nature, lasting some weeks or months, with very characteristic remissions and exacerbations; or finally, but chiefly in older children, recovery may occur, although with paralytic symptoms perhaps remaining. The prognosis is on the whole unfavorable; as far as the difficulty in diagnosis permits of the drawing of any conclusions.

Diagnosis.—It is only at autopsy that diagnosis can be at all certain. The development of symptoms of effusion of blood is liable to be more rapid than in epidural hemorrhage, but not sufficiently so to give much aid in distinguishing between the two disorders. Meningitis is slower of onset, and has higher fever with less tendency to localizing symptoms. Lumbar puncture gives a clear fluid, or one reddish or discolored, or even distinctly blood-stained.

Treatment.—This can be merely palliative and symptomatic. Ice to the head and free purgation are to be used to relieve the intracranial tension, and lumbar puncture or that of the cranium may be tried for the same purpose.

LEPTOMENINGEAL HEMORRHAGE

Etiology and Pathological Anatomy.—By far the most common cause is trauma of some sort. Consequently the disease is especially frequent in the new born, as a result of the continued pressure of a difficult and prolonged labor; much less often produced by the employment of instruments. Wilson[1] observed meningeal hemorrhage in 15 out of 20 autopsies in infants dying in the first 3 weeks of life; Wehije[2] in 959 autopsies in new-born infants found death due to intracranial hemorrhage in 122 instances, this being in the brain-substance in only 36; and Hedren[1] noted 65 cases of intracranial hemorrhage, involving the meninges in 90.7 per cent., in 700 infant cadavers. In 50 of the cases the labor had been spontaneous. That it happens so readily at this period depends, too, upon the extremely delicate structure of the pial vessels. Much less often meningeal hemorrhage is produced by trauma later in childhood, or occurs as a result of some infectious disease, such as measles, variola, scarlet fever, diphtheria, or pneumonia; or may develop during a hemorrhagic disorder. Hemorrhage may be a secondary lesion to intense congestion attending a convulsion or a paroxysm of pertussis; or sometimes to embolism or thrombosis of the cerebral vessels or, oftener, the sinuses. Generally the hemorrhage is confined to the region below the arachnoid, but it is possible in occasional instances for it to burst through this and produce a complicating subdural hemorrhage. It may be only punctiform, or a much larger amount of blood may occupy the arachnoidal space. The effusion may be either superficial and cover a considerable area over the convexity of the brain, or may be at the base. In the latter situation it is chiefly in the posterior fossa beneath the tentorium. In the former it is usually bilateral and most marked near the median fissure, although not equally extensive on both sides (Fig. 334). McNutt[2] found basal hemorrhage oftenest in head-presentations, and hemorrhage over the convexity most frequent in breech or foot presentations. This would not appear to be a uniform experience. The later secondary changes consist of cysts, meningo-encephalitis, sclerosis and atrophy, and secondary degeneration in the lateral tracts of the spinal cord.

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[1] Phila. Med. Journ., 1901, I, 226.

[2] Ref., Sloan, Cleveland Med. Journ., 1915, XIV, 808.

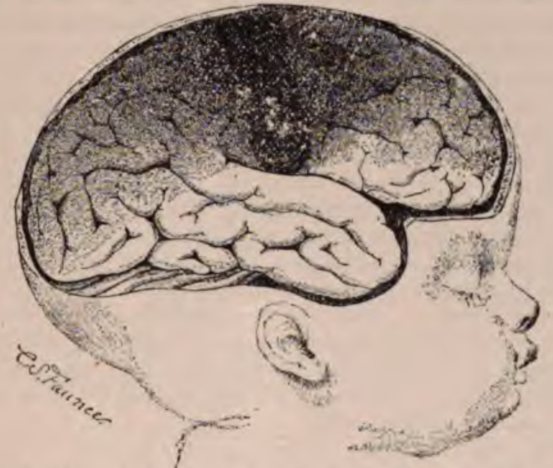

FIG. 334.—MENINGEAL HEMORRHAGE IN THE NEWBORN. (*McNutt, Amer. Journ. of Obstetrics*, 1885, *XVIII*, 76.)

Symptoms.—In cases of hemorrhage of very small size occurring at birth there are generally no symptoms either immediate or later. Larger hemorrhages, too, may produce at the time no characteristic symptoms, and the infants merely are asphyxiated, atelectatic and comatose very promptly after birth; or become so a day or two later. In other instances there are the distinct signs of increased intracranial pressure, with unconsciousness, slow feeble pulse, irregular superficial respiration, and bulging of the anterior fontanelle. Evidences of irritation, as represented by convulsions, are of very frequent occurrence, and are usually bilateral. Paralysis may also be present, although less often among the earliest manifestations. The later symptoms in cases which survive will be discussed under Infantile Cerebral Paralysis (p. 366).

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[1] Sevenska Läkaresällskapets Handlinger, 1918, XLIV, 53. Ref. Journ. Amer. Med. Assoc., 1918, LXX, 1988.

[2] Amer. Journ. Med. Sci., 1885, LXXXIX, 58; Amer. Journ. Obstet., 1885, XVIII, 73.

Course and Prognosis.—The prognosis is very uncertain, and serious in all cases except where the lesion consists of punctiform hemorrhages. These are of little importance, and are absorbed leaving no trace. Small effusions of somewhat greater size than these may give rise to no immediate symptoms, but may in later years show the evidence of damage done. Hemorrhage of decidedly large amount may produce death in a few hours or days; and if the infant escapes with his life, paralysis, epilepsy and idiocy, separately or combined, are liable to develop later. Sometimes the lesion becomes infected by germs and suppuration in the effused blood follows. I have seen a very large amount of pus produced in this way.

Diagnosis.—This is to be based on the symptoms of intracranial pressure and the results of lumbar puncture. By the latter a bloody fluid will be obtained which exhibits partly degenerated blood-corpuscles. The history of paralytic symptoms is of more aid than is that of general convulsions in forming a diagnosis, since the latter readily occur in so many other conditions. Localized convulsions are of greater import. From *subdural hemorrhage* the disease is to be distinguished by the usually wider spread of the effusion with consequent less marked localization of the symptoms. There is also less decidedly progressive loss of function of an entire hemisphere.

Treatment.—Little can be done in the line of medical treatment once the hemorrhage has occurred, except the obtaining of rest for the brain by the allaying of all nervous manifestations through the employment of sedative drugs. Operative interference has been performed in some cases with good results (Cushing)[1] and others, and is to be urged in cases where the diagnosis can be made with sufficient accuracy, which unfortunately is often not the case. Of most importance is the attempt to prevent hemorrhage by not permitting parturition to be unduly prolonged.

MENINGISMUS

(Pseudomeningitis)

In this connection reference may be made to the condition, first described under this title by Dupré,[2] occurring in the course of different affections, especially the acute infectious fevers, pneumonia, and marantic states in infancy, and characterized by symptoms resembling those of meningitis, but due entirely to a functional disturbance produced by toxins or in other ways. No pathological lesions can be found post-mortem, and the spinal fluid shows no evidence whatever of inflammation. It is this latter especially which would differentiate the disorder from serous meningitis. Most writers are disposed to abandon the term;

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[1] Amer. Journ. Med. Sci., 1905, CXXX, 563.

[2] Congress de Lyon, 1895, and Traité des mal. de l'enf., Grancher, 1905, IV, 27.

and it is certain that many, and probably the majority, of the cases are in reality serous meningitis. It is, however, a very convenient appelation for a symptom-complex which is of frequent occurrence, and which it seems difficult to attribute to meningitis of any form in many instances. The prognosis, apart from that of the causative disease, is favorable if the diagnosis is certain, and no special treatment is required.

LEPTOMENINGITIS

Several forms of this disease are to be considered, these being (1) Serous meningitis; (2) Acute purulent meningitis, one form of which, Cerebrospinal fever, has already been discussed; and (3) Tuberculous meningitis.

SEROUS MENINGITIS

(Sero-fibrinous Meningitis; Ependymitis; Serous Apoplexy; Acute Acquired Hydrocephalus)

Concerning the nature of this malady there was formerly much discussion. Its existence as an entity is now admitted. The title applies properly to acute cases with meningeal symptoms in which the cerebrospinal fluid is increased in amount, but exhibits a normal or but a slight increase of the number of leucocytes; while microörganisms are not discoverable in it, or are present in inconsiderable numbers.

As the name implies, the fluid is an exudate of an inflammatory nature and not a mere transudate. The occurrence of this disease with comparative frequency was first brought into prominence by Quincke[1] and the title applied to it by him.

Etiology.—The disease is liable to develop especially in the first 2 years of life, although not confined to this period. It is almost always a secondary one; not infrequently to otitis media or other neighboring purulent processes, the effusion usually being the result of the action of toxins rather than of transmitted infection. Among other causes are trauma involving the head, yet without suppuration; syphilis; and, finally, various acute infectious diseases, as typhoid fever, gastroenteritis, measles, whooping-cough and particularly pneumonia.

Pathological Anatomy.—In autopsies made early in the attack there is found a circumscribed or often diffuse edematous swelling of the meninges, together with effusion of fluid. The process may involve only the cortex or base, or the ventricles as well. If the case has lasted some time there may be dilatation of the ventricles and flattening of the convolutions, and in some instances there is evidence of a circumscribed meningitis. The fluid as discovered at autopsy, or as obtained by lumbar or cranial puncture, is perhaps at first moderately increased in amount, and later to a greater degree and under pressure; and is entirely clear. It exhibits a slight increase of protein and of the mononuclear cells; the number which might be considered significant usually being not under 10 or 12 to the c. mm. (See Cerebrospinal Fluid, p. 234.) A delicate fibrin clot may form on standing. As already stated, bacteria are absent or found in very small numbers. Microscopical examination of the meninges shows a certain amount of cellular infiltration.

Symptoms.—The symptoms are subject to considerable variation, depending upon the severity of the attack, the portion of the meninges chiefly involved, and the nature of the disease to which the condition may be secondary. In the most acute cases, sometimes called *serous apoplexy*, the disease begins with high fever and sometimes even hyperpyrexia; severe repeated convulsions; contracted pupils; possibly slight stiffness of the neck, and coma. There is nothing positively characteristic of meningitis. Much more frequently the development is more gradual and the course less intense. The onset is then with fever of moderate degree; headache; stiffness of the neck; rigidity of other regions; sometimes vomiting; often delirium; grinding of the teeth; convulsions

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[1] Volkmann's Sammlung klin. Vorträge, 1893, Inner. Med., No. 23.

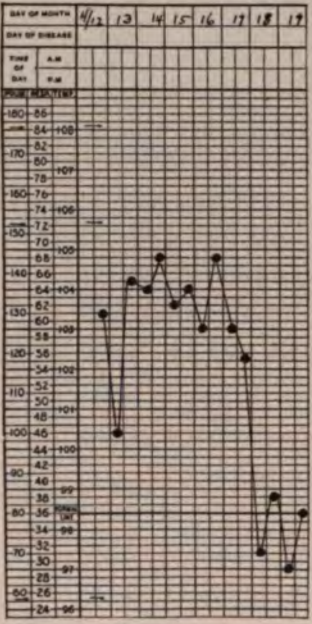

FIG. 335.

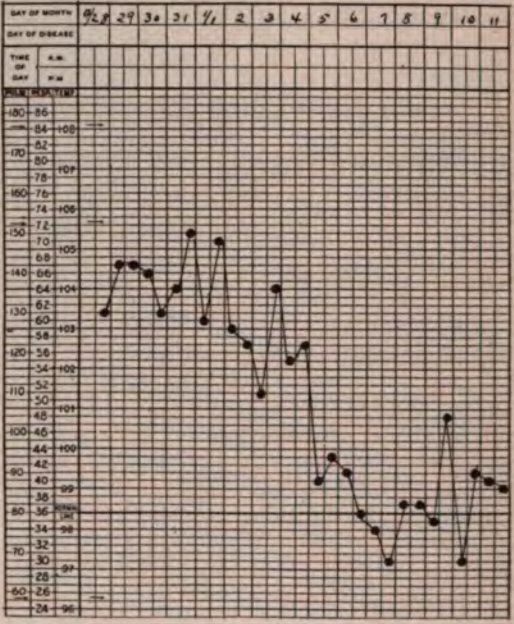

FIG. 336.

FIG. 335.—SEROUS MENINGITIS WITH CELL-COUNT NOT ABNORMAL BUT PRESSURE INCREASED.

Antonio DeM., aged 28 months. Admitted to Children's Hospital, Apr. 12. Said to have developed pneumonia 8 days before. Rigidity of body began 3 days ago, first in legs, and followed by opisthotonos. Diarrhea last few days. While in hospital respiration from 30 to 60+, pulse 110 to 160, restless, coughing, lesions of pneumonia found in lungs, body and limbs very rigid, general symptoms those of meningitis; Apr. 13, spinal puncture gave 12 c.c. (0.41 fl.oz.) of clear, watery fluid under increased pressure, no organisms, 7 leucocytes to the c.mm.; Apr. 14, stiffness still marked, knee-jerks exaggerated, tâche very marked, lesions of pneumonia still well-developed; Apr. 15, condition poor, pulse rapid, comatose, vasomotor flushing and paling as in meningitis; Apr. 18, critical fall of temperature; Apr. 19, rigidity has disappeared, lungs clear, knee-jerks normal, examination of lungs practically negative. Recovery.

FIG. 336.—SEROUS MENINGITIS, CELL-COUNT SUGGESTING TUBERCULOUS MENINGITIS.

Miller P., 11 months old. Admitted to the Children's Hospital of Philadelphia, Dec. 28, 1914. Had been ill apparently a few days with anorexia, vomiting and drowsiness. Examination showed some râles in chest. Dec. 30, ears normal, quite pale, quiet, apathetic, marked tâche, slight retraction of abdomen, nothing definite in lungs, leucocytes 35,800; Jan. 5, distinct pneumonic consolidation of left lung, and general evidences of meningitis persisting; Jan. 11, improving, still some rigidity of neck. Complete recovery. Lumbar puncture on several occasions showed: Jan. 2, distinct increase of fluid, 190 cells to the c. mm.; Jan. 4, numerous cells with predominating lymphocytes, no microörganisms found; Jan. 9, lymphocytes and polymorphonuclear cells about equal number; Jan. 11, dry tap.

or a tendency to coma; and evidences of intracranial pressure. The convulsions may be general, unilateral, or localized in other ways. The fever attending may be the result chiefly of the primary disease, as, for instance, in the serous meningitis occurring in the course of a pneumonia (Figs. 335; 336). It is especially, however, in ventricular serous meningitis (*acute acquired internal hydrocephalus*) that symptoms of very decided intracranial pressure develop, such as choked discs; slow irregular pulse; and, in infancy, great bulging of the anterior fontanelle, either with convulsions or coma. In some forms of serous meningitis the onset is so gradual that the case greatly resembles tuberculous meningitis.

Course and Prognosis.—The violent, acute cases progress rapidly, and usually end in death in a few hours up to a few days; but it is possible even for these to take a sudden turn for the better, and then either to run an ordinary course or to recover quickly. In those of a less violent form the symptoms often disappear rapidly, either with the subsidence of an acute infectious disease, or as the result of operation upon some neighboring purulent focus, or spontaneously without this. Other instances go on to a fatal ending after days or weeks; and still others pass into the condition of chronic acquired internal hydrocephalus, which is attended in the severer cases by optic atrophy, paralysis of some of the cranial nerves, and a spastic condition of the limbs; the symptoms being now dependent upon pressure rather than upon inflammation. A few such are due to syphilis. A similar final condition is seen in the chronic stage of cerebrospinal fever (Vol. I, p. 425).

The prognosis is on the whole favorable, and the majority of cases will recover. In 80 cases studied by Dubois and Neal[1] there were 22 deaths, and these generally from the primary disease. Many cases which begin severely and appear to be certainly either purulent or tuberculous in nature, are in reality instances of serous meningitis and do well. It is, however, of quite frequent occurrence that cases in which the life has been saved later exhibit conditions dependent upon the earlier inflammation; such as epilepsy, or a slight mental defect or other psychic disturbance; or there may be found a tendency to recurrence of the meningitis in after years.

Diagnosis.—The diagnosis is often of the greatest difficulty. It rests upon the obtaining by lumbar puncture of a clear fluid under slight pressure, with fibrin and an increase of protein, but with very few or no germs, and only a moderate increase of the cells, these being of a mononuclear type. The fluid of *tuberculous meningitis*, however, although usually exhibiting a slight turbidity and a somewhat greater number of cells (see pp. 234, 333), may in many cases be very like that of serous meningitis in character, there being no turbidity whatever. In both disorders the cells are of the lymphoid type. Only repeated examination may show the presence of tubercle bacilli, and sometimes inoculation of a guinea-pig is required to demonstrate this. The chief diagnostic difference from tuberculous meningitis is the more rapid onset, the shorter course, and the absence of the indefinite prodromal symptoms which characterize the latter disease.

Purulent meningitis of any variety is more easily recognized, since polymorphonuclear leucocytes are found in abundance, generally with numerous bacteria. It is to be noted, however, that exceptionally in the earlier stages of cerebrospinal fever, and commonly in its later stages, the spinal fluid may be clear. The meningitic type of *poliomyelitis* may simulate serous meningitis, and the lumbar puncture may reveal a spinal fluid which is identical in character with that of the latter. The prompt development of characteristic paralysis will remove all doubt. The diagnosis from *cerebral abscess* will be considered under that heading. Finally the distinction is to be made from *meningismus*, if we allow that a difference in fact exists, the chief diagnostic symptom being that in this latter condition there is no sign whatever in the lumbar fluid of the presence of any meningeal inflammation.

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[1] Amer. Jour. Dis. Child., 1915, IX, 1.

Treatment.—The most efficacious treatment is prompt and repeated lumbar puncture, which often gives decided relief. Since the disease is secondary, efforts should be made to locate and remove the original cause, and to sustain life and relieve symptoms until this can be accomplished. For this purpose absolute rest and quiet are to be sought, warm baths given for the reduction of temperature or the relief of nervous manifestations, and ice-bags applied to the head. Mercurial inunctions should be tried even when syphilis is not known to be present. Headache and other troublesome symptoms are to be relieved by suitable medication.

ACUTE PURULENT LEPTOMENINGITIS

(Meningitis Simplex)

That variety of purulent leptomeningitis dependent upon the meningococcus possesses peculiarities in sufficient number to warrant the separate consideration which has been given it. (See Cerebrospinal Fever, Vol. I, p. 415.) To the remaining forms the title *Meningitis simplex* is sometimes applied.

Etiology.—The germs capable of producing the disease are of considerable variety. Among them are to be mentioned especially the pneumococcus, streptococcus, staphylococcus, influenza bacillus, diplobacillus pneumoniæ of Friedländer, typhoid bacillus, bacillus pyocyaneus, colon bacillus, and gonococcus. In addition acute meningitis is sometimes dependent upon syphilis, and has repeatedly been observed in the course of mumps. Dopter[1] saw 158 cases of meningitis occurring as a complication in 1705 patients with the latter disease. These should possibly be classed under serous meningitis. Syphilitic meningitis is generally of a chronic nature. As to the relative frequency of the different forms of leptomeningitis: in 197 cases under 3 years of age, analyzed by Holt,[2] there were 138 tuberculous, 22 pneumococcic, 24 meningococcic (sporadic cases), 4 influenzal, 10 exhibiting the staphylococcus or streptococcus, and 1 the colon bacillus. In 2 of the cases there was a mixed infection. The majority of the instances of meningitis occur in infancy, except perhaps in the case of septic meningitis.

Clinical Forms.—A few of the clinical forms dependent upon the different germs which may be found require brief descriptions:

Pneumococcic Meningitis.—This is perhaps the most frequent variety next to that due to the diplococcus intracellularis already described; always, however, with the exclusion of tuberculous meningitis, which much exceeds simple purulent meningitis of any sort in frequency, except during the epidemic prevalence of cerebrospinal fever. Of 119 cases of meningitis in the wards of the Children's Hospital of Philadelphia during 5 years, 6 were proven to be pneumococcic. The disease may appear to be primary, at least so far as clinical evidence of pneu-

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[1] Paris méd., 1910, I, 35.

[2] Amer. Jour. Dis. Child., 1911, I, 26.

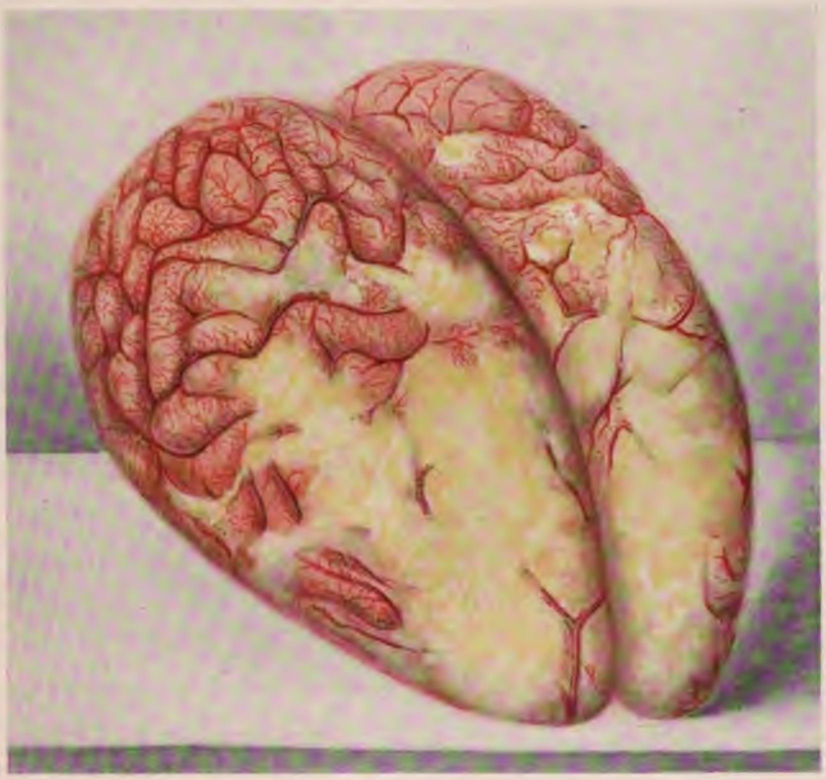

FIG. 337.—PNEUMOCOCCIC MENINGITIS.

Infant of 7 months. Vague nervous manifestations followed by symptoms of pneumonia, otitis media, and mastoiditis. Autopsy showed a thin layer of lymph over a portion of the cortex and at the base. Pneumococci found, with staphylococci and streptococci probably as secondary development.

mococcus infection elsewhere is concerned. Sometimes the affection is actually primary, the pneumococci having penetrated to the meninges through the ethmoid bone; while in other instances there is an acute pneumococcic inflammatory focus primary elsewhere in the body, although undiscovered during life. In the majority of cases, however, the meningitis is clinically secondary to infection elsewhere, oftenest in the lungs or pleura; sometimes in the pericardium or peritoneum, or in an infected joint. Yet the number of occasions on which a pneumococcic meningitis develops in connection with pneumonia is relatively very small. Thus in 1050 cases of pneumonia studied by Rolly,[1] only 6 instances of pneumococcic meningitis were seen.

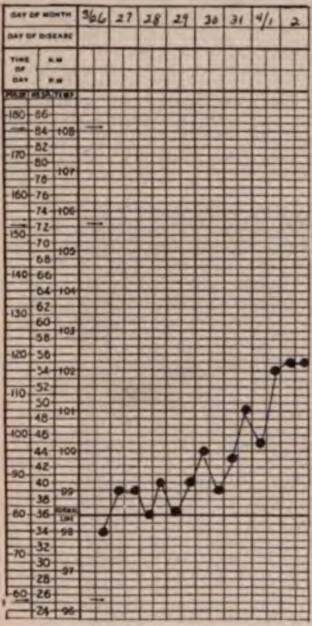

FIG. 338.

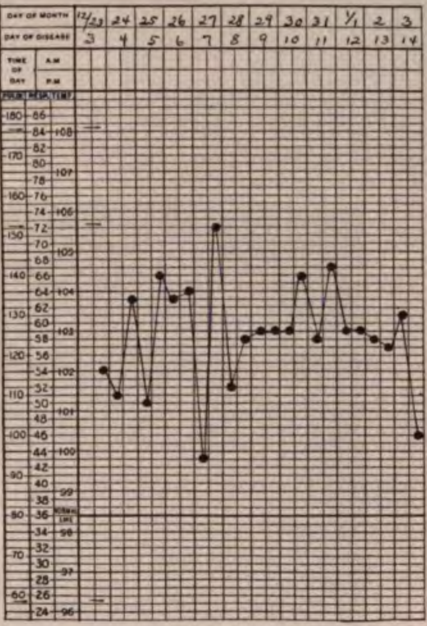

FIG. 339.

FIG. 338.—PNEUMOCOCCIC MENINGITIS, WITH SLOW COURSE AND LITTLE FEVER.

David M., 8 months old. Admitted to the Children's Ward of the Hospital of the University of Pennsylvania, Mar. 6. Began to vomit 3 weeks before, became apathetic, ceased to play, and developed retraction of the head; said to have been fever at first, but disappeared afterward. 2 days before admission grew worse, became stuporous. Vomiting stopped soon after the onset. While in hospital remained stuporous most of the time, markedly retracted head, well-developed tâche, child cried if head was moved, anterior fontanelle bulging, thick yellow discharge from eyes and nose, apparently no rigidity of the extremities. Lumbar puncture done on several occasions, fluid under pressure and showed the pneumococcus, serum injected intravenously and again intraspinally. Condition grew worse and died on Apr. 3. Fluid from spinal column showed 2600 leucocytes per c. mm., 96 per cent. being polymorphonuclear cells.

FIG. 339.—PNEUMOCOCCIC MENINGITIS, HIGH FEVER.

Lillian S., 10 months old. Admitted to Children's Hospital, Dec. 23, 1915. Taken ill Dec. 22 with convulsions. These continued more or less. No positive symptoms of pneumonia were found, but respiration was from 50 to 70 per minute, and lumbar puncture showed slightly cloudy fluid, with numerous polymorphonuclear cells, globulin ++, pneumococci found. Died Jan. 3, 1916.

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[1] Deut. med. Woch., 1911, XXXVII, 774.

Pathological Anatomy.—The lesions are very similar to those observed in cerebrospinal fever. There is, however, often a greater development of fibrinopurulent exudate, especially on the convexity of the brain, although the base is involved to a less degree than in cerebrospinal fever, and sometimes not at all. The ventricles may sometimes be even more affected than the surface, but the reverse is oftener the case (Fig. 337). Involvement of the spinal cord is usually slight or absent.

Symptoms.—These are largely the manifestations characteristic of all forms of purulent meningitis. (See Cerebrospinal Fever, Vol. I, p. 418.) In the cases developing in children previously entirely well, or at least not severely ill with other affections, there occurs an abrupt onset, with high fever, headache, vomiting, very frequently convulsions, delirium, and stupor (Figs. 338; 339). Stiffness of the neck, retraction of the head, opisthotonos, and hyperesthesia are not as common as in cerebrospinal fever or tuberculous meningitis. This is dependent upon the lesser frequency and degree of involvement of the base as compared with the convexity of the brain. Very often in subjects seriously ill with other diseases pneumococcic meningitis may run an entirely latent course, the diagnosis being made only at autopsy.

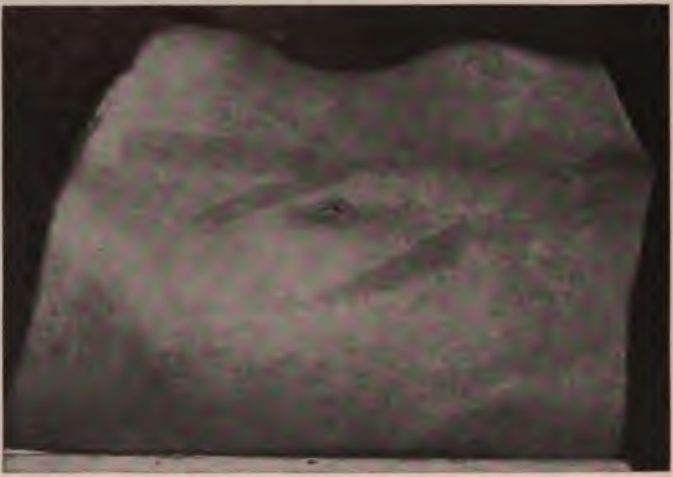

FIG. 340.—TACHE MENINGITIQUE.

From a pâtient, aged 4 years, suffering from cerebrospinal fever, in the Children's Ward of the Hospital of the University of Pennsylvania. The broad red lines are shown black in the photograph.

There are a number of special symptoms sometimes present in this as in all forms of meningitis to which reference may be made here. The knee-jerks are often exaggerated, especially early in the attack. Later they may be lost. Ankle-clonus is common. Kernig's sign[1] consists in the inability to extend the leg on the thigh by passive movement while the thigh is flexed at a right angle upon the trunk. It is very frequently present but by no means always so; and the indisposition of children under any circumstances to permit of the procedure renders it, in my experience, of little value. The "neck-phenomenon," or Brudzinski's sign,[2] consists in a flexion of both of the lower extremities at the hip and knee when the neck is passively bent forward. It is nearly always present, and is absent in other conditions. The failure to obtain it does not exclude meningitis. The "contra-lateral reflex," described earlier by the same writer[1] is shown in the drawing up of one leg if the other is passively flexed at the hip-joint. This is less constantly present than the previous sign. The "tâche cerebral" (Fig. 340), to which reference has been made in discussing cerebrospinal fever (Vol. I, p. 423), consists of a broad, slowly appearing and long-continuing red line developing in the position where the finger-nail has been drawn over the skin. It is usually present in all forms of meningitis and is very suggestive when combined with other symptoms, but by no means conclusive, inasmuch as it may sometimes be found in other conditions.

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[1] Berl. klin. Woch., 1884, XXI, 829.

[2] Arch. de méd. des enf., 1909, XII, 745.

Course and Prognosis.—The course is short and usually severe, the attack lasting seldom more than 3 or 4 days. Cases of recovery have been reported, as for instance by Rolly,[2] Cumming,[3] Brown,[4] and others; but are very exceptional.

Diagnosis.—The apparently primary cases cannot be certainly distinguished from other forms of severe acute purulent leptomeningitis except by lumbar puncture. This gives a turbid fluid in which pneumococci can readily be recognized, together with numerous polymorphonuclear cells. Yet even without lumbar puncture the existence of a pneumococci meningitis is to be suspected when, during the course of pneumonia, empyema, pericarditis, peritonitis or otitis, the symptoms of meningitis suddenly develop. It is to be remembered, however, that convulsions, vomiting, and other meningitic symptoms, which often occur during pneumonia and frequently last for some days, are not to be regarded as evidences of the existence of pneumococcic meningitic. Much more frequently such manifestations are produced by serous meningitis, or are merely toxic in nature. (See Cerebral Pneumonia, p. 80.) Lumbar puncture and the examination of the fluid is required. The diagnosis of pneumococcic and other forms of simple purulent meningitis from the tuberculous variety will be considered in discussing the latter disease (p. 335).

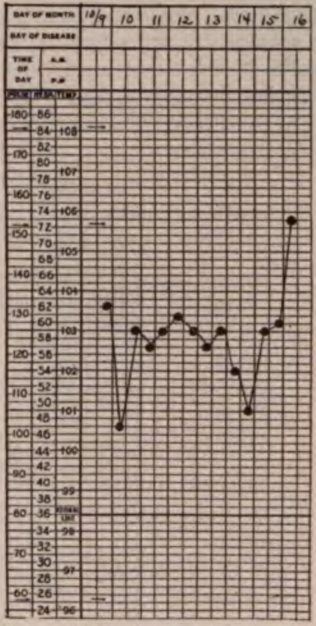

FIG. 341.—SEPTIC (STAPHYLOCOCCIC) MENINGITIS.

Thomas W., aged 12 years. Entered Children's Ward of University Hospital, Oct. 9. About 2 weeks previously developed diffuse pain in left leg, with fever, then on Oct. 7 in the left arm, and became delirious. Examination showed no positive localization in the limbs, slight dry cough, condition supposed to be typhoid fever with neuritis, grew steadily worse, very hypersensitive all over, rigidity of neck, evident signs of meningitis. Lumbar puncture on Oct. 13 gave fluid under pressure, containing staphylococcus pyogenes citreus; Oct. 14, delirious, unconscious, papulo-vesicular eruption over the body, some places in the form of blebs. Fluid obtained today was turbid, under high pressure, cell-content 98 per cent. polymorphonuclears and 2 per cent. lymphocytes. Died Oct. 16.

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[1] Wien. klin. Woch., 1908, XXI, 255.

[2] *Loc. cit.*

[3] Lancet, 1912, II, 294.

[4] Lancet, 1916, II, 519.

SEPTIC MENINGITIS (Fig. 341).—Here are to be included the cases depending upon the action of pyogenic germs, such as the staphylococcus and streptococcus of different species. In the new born the disease is associated oftenest with umbilical infection; in older subjects with otitis, empyema, erysipelas, and suppuration of the joints due to other causes than the pneumococcus or the tubercle bacillus. The **lesions** are very similar to those of pneumococcic meningitis, although with the production of a smaller amount of fibrin; and the **clinical features**, too, are like those of this variety. The onset is usually sudden, and the symptoms well-marked except in cases already greatly ill with septic involvement in other regions, in which event the cerebral indications may be poorly defined and readily overlooked. The course is short and the **prognosis** almost entirely unfavorable. Dubois and Neal[1] could find but 3 reported cases of recovery to which they added a 4th. Leighton and Pringle[2] also report 2 instances of recovery. The **diagnosis** is to be made by lumbar puncture, which gives a turbid fluid with many polymorphonuclear cells, and shows the presence of some of the pyogenic microörganisms mentioned. The diagnosis is especially difficult between a localized septic meningitis from otitis, and an abscess of the brain.

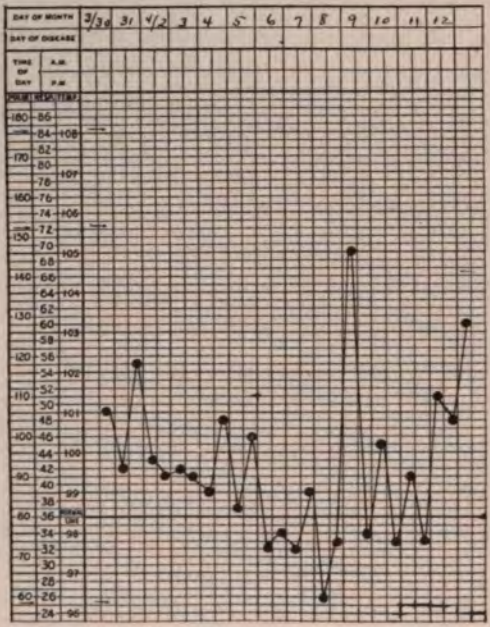

FIG. 342.—FATAL INFLUENZAL MENINGITIS.

Samuel M., aged 2 years. Entered the Children's Hospital of Philadelphia, Mar. 30. Earache 2 weeks previously, ear been discharging for 2 days. Promptly developed signs of meningitis with restlessness and deviation of the eyes; Apr. 3, lumbar puncture gave cloudy spinal fluid which yielded a pure culture of the influenza-bacillus, 70 leucocytes to the c. mm.; Apr. 7, condition unimproved, intraspinal injection of influenza-serum given yesterday and again on this date; Apr. 8, lumbar puncture showed very cloudy fluid. Died Apr. 12.

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[1] Arch. of Ped., 1915, XXXII, 28.

[2] Journ. Amer. Med. Assoc., 1915, LXIV, 2054.

The condition of the spinal fluid is the chief guide; since this is generally clear in cases of abscess. In addition there is in abscess more headache and a greater degree of optic neuritis.

INFLUENZAL MENINGITIS.—This is an uncommon condition which has been studied by Adams,[1] Wollstein,[2] Torrey,[3] and others. Wollstein collected 58 recorded instances in which the bacillus of Pfeiffer was reported present in pure culture or with other germs; 8 doubtful cases are not included here. More than half of the cases of the series collected occurred in the 1st year of life. Torrey added 28 additional reported cases, and others have since been recorded. With a greater certainty in the recognition of the influenza bacillus, the affection may be found to be more common than supposed. The disease is usually secondary to manifestations of influenza elsewhere in the body. The **lesions** and the **symptoms** are similar to those of other forms of purulent meningitis (Figs. 342; 343). Lumbar puncture gives a turbid or a distinctly purulent fluid with numerous polymorphonuclear cells, and with microörganisms, both extra- and intracellular, generally not in large numbers. It is only by the cultural study of the spinal fluid that a diagnosis can be made with certainty. The **prognosis** is unfavorable, although less so than in the other varieties described; 5 of Wollstein's series having recovered. Torrey's statistics raise the number to 7.

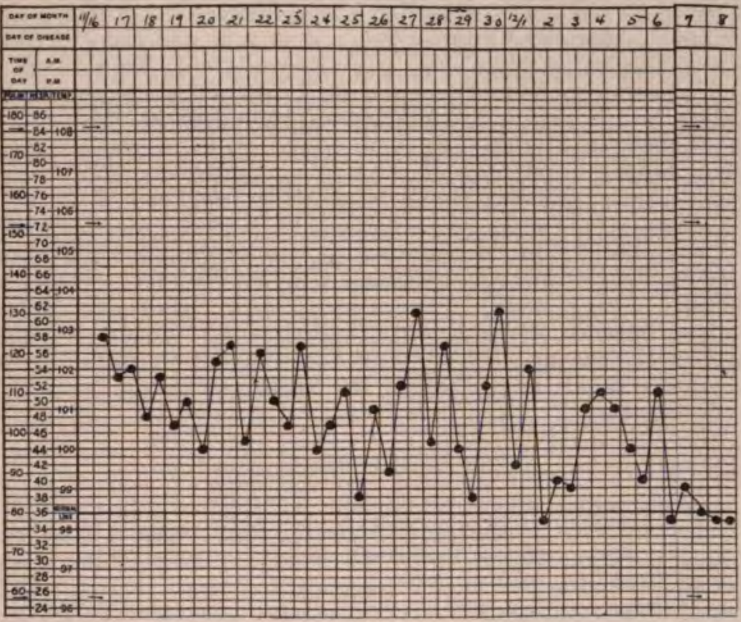

FIG. 343.—INFLUENZAL MENINGITIS. RECOVERY.

Girl, 11 years old. Taken ill Nov. 15, with symptoms of influenza. Evidences of meningitis developed on Nov. 17, and lumbar puncture revealed turbid fluid with influenza-bacilli. Puncture repeated on a number of occasions and injections of Wollstein's serum given. Decided improvement began by Nov. 28, and recovery was finally complete. *Courtesy of Dr. R. G. Torrey.*

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[1] Arch. of Pediat., 1907, XXIV, 721.
[2] Amer. Jour. Dis. Child., 1911, I, 42.
[3] Amer. Journ. Med. Sci., 1916, CLII, 403.

Meningitis Due to Other Microörganisms.—Of these may be mentioned especially that dependent upon the typhoid bacillus, the presence of which has been reported in a number of instances collected by Cole.[1] Infection with the colon bacillus has also occurred, and in very rare instances with the gonococcus. The streptothrix was observed in a case reported by Rutelli[2] and the bacillus proteus in one by Göbel.[3]

Treatment of Simple Purulent Leptomeningitis.—There is little to be said in this connection. In nearly all instances the only treatment possible is symptomatic. The child should be kept quiet in a somewhat darkened room away from noise. All unnecessary moving of the body is to be avoided on account of the pain and nervous disturbance which this may produce. Attention is to be paid to the giving of suitable nourishment. Repeated lumbar puncture may be performed in the effort to relieve cerebral compression, and is certainly to be recommended, especially in cases of influenzal meningitis, where there is greater chance of recovery. It has been advised to administer large doses of hexamethylenamine by the mouth, or inject it into the spinal canal after lumbar puncture, in the hope of checking the multiplication of the germs. For intraspinal administration 10 to 15 grains (0.65 to 0.97) of the drug may be dissolved in sterile water, and all or a portion of it injected at one time. The solution may be kept in ampoules until needed. Beyond this little can be done except to relieve pain and nervous symptoms by appropriate medication. Wollstein[4] produced a serum which, when given intraspinally, checked the progress of experimental influenzal meningitis. In cases of the pneumococcic type the injection of an antipneumococcic serum should be tried, selecting the strain of pneumococcus which the case exhibits.

TUBERCULOUS MENINGITIS

Although undoubtedly a disease long existing and mentioned by other writers before Whytt,[5] this author was the first to give a clear satisfactory description of it. It is by all odds the most frequent form of leptomeningitis occurring in children, equalling more than all the others combined, if the epidemic outbreaks of cerebrospinal fever be disregarded. Of 239 cases of meningitis occurring in the Children's Hospital of Philadelphia during a period of years, during some of which cerebrospinal fever in an epidemic form was present in the city, 121 were diagnosed as tuberculous, and in probably the majority of these the diagnosis was confirmed by autopsy or by lumbar puncture. The influence of the disease upon the death-rate in early life is very decided. Méry and Armand-Delille[6] estimate that 5 per cent. of the deaths among children in Paris are due to it.

Etiology.—Inheritance and family relationship play the *predisposing* part seen in all tuberculous affections. There is undoubtedly a marked inherited tendency; yet the degree of this is not clear in the presence of the coexisting much greater probability of direct infection from tuberculous members of the family or in other ways. The age of the child is a matter of moment. It is generally maintained that the period from the 2d to the 6th year inclusive exhibits the greatest number of cases; the majority of these being in the 2d and 3d years. Yet a large number occur in the 1st year. From 6 to 10 years the cases are less numerous, and very few are encountered after the age of 10 years. In a study of 351 cases by H. Koch[1] 68 per cent. were seen in the first 4 years, and only 3.7 per cent. in subjects from 10 to 14 years of age. The greatest number were observed in the 2d year. Those seen in the 1st year of life are nearly always in its latter half. It may be mentioned, however, that Rilliet and Barthez[2] saw 1 at 5 months; Reich[3] reported 7 of not over 3½ months; Herter[4] in 24 cases found 6 under 8 months of age; and Holt[5] had 3 under 3 months in 218 cases of tuberculous meningitis.

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[1] Johns Hopk. Hosp. Rep., 1904, XII, 379.
[2] La Pediatria, 1915, XXII, 713.
[3] Deut. Archiv f. klin. Med., 1914, CXVI, 119.
[4] Jour. Exper. Med., 1911, XIV, 73.
[5] Observations on the Dropsy of the Brain, Edinburgh, 1768.
[6] Traité des mal. de l'enf., Grancher, 1905, IV, 71.

Sex appears to have no certain influence. Season seems to be an etiologically predisposing factor, the largest number of cases being observed in the spring; but this would appear to be due to the greater confinement to the house during the winter season, or the tendency of respiratory diseases to occur in winter and spring, and the consequent acquiring or awakening to activity of a focus of tuberculous infection somewhere in the body. Acting in the same way, many other diseases, and particularly measles, pertussis and typhoid fever, are liable to be followed by tuberculous meningitis. Trauma, especially of the head, has seemed to be a predisposing cause in a number of reported instances.

The *direct cause* is, of course, infection by the tubercle bacillus. At what time this has taken place is always uncertain, for tuberculous meningitis is always pathologically a localization secondary to a lesion in some other part of the body. This lesion may be a recent one, as in cases closely following upon a tuberculous bronchopneumonia; or may date back indefinitely, the time of its development not being discoverable, and no symptoms of tuberculosis ever having shown themselves.

Clinically, however, in the majority of cases tuberculous meningitis appears to be a primary disorder, the bacilli having been transported to the brain by way of the lymph or often the blood from some small unrecognized focus, very frequently in the lungs or especially in the tracheobronchial lymphatic glands. In 67 autopsies on tuberculous meningitis reported by Haushalter and Fruhinsholz[6] involvement of these glands was found in 63 instances. Not infrequently it is the terminal process of a general tuberculosis, the symptoms of which had been of the usual vague nature, and the diagnosis often questionable or wrongly made. In older children it is not uncommon to see tuberculosis meningitis develop as a sequal to an active or a healed tuberculous process in the bones or joints. It does not follow, however, that a meningitis must be tuberculous because evidences of tuberculosis are found elsewhere in the body; and, on the other hand, it is to be remembered that tuberculosis meningitis may complicate other affections not of this nature.

The method by which the primary infection has taken place has already been discussed. (See Tuberculosis, Vol. I, p. 543.) Often in comparatively isolated districts this can be traced directly to association of the child with affected individuals in the neighborhood. In larger cities

[1] Zeitschr. f. Kinderheilk., 1912, V, 355.
[2] Sanné, Mal. des enfants, 1891, III, 1027.
[3] Berl. klin. Woch., 1878, XV, 551.
[4] New York Med. Jour., 1901, LIII, 42.
[5] Amer. Jour. Dis. Child., 1911, I, 26.
[6] Archiv de méd. des enf., 1902, V, 157.

where the tubercle bacilli are naturally so wide spread, it is often impossible to discover in any way the source of infection. In nearly all instances it is the human type of the bacillus which is the active agent.

Pathological Anatomy.—The process in brief consists of the formation of miliary tubercles together with an inflammatory exudate. The dura is tense, the convolutions flattened, and the cavity of the arachnoid and the ventricles more or less filled with serofibrinous or fibrinopurulent exudate; the presence of pus perhaps depending upon a mixed infection. The amount of fibrin and of pus is usually small as compared with other forms of meningitis, and the fluid is clear or slightly cloudy. Tubercle bacilli are found in the fluid, and not infrequently other germs are present in addition (Rhein).[1] A part of the effusion is of an edematous nature, but the actual tuberculous exudation, especially at the base, is sticky and adheres closely to the meninges. The pia mater at the base, and to a less extent on the convexity, is slightly cloudy, injected, and exhibits more or less numerous miliary and submiliary tubercles, often with tuberculous nodules of larger size. Tubercles are often numerous, too, in the Sylvian fissure. The membranes about the base, optic chiasm, cranial nerves, and the Sylvian fissure are often much matted together and thickened. Tubercles and leucocytic infiltration are also found on the lining of the ventricles, and to some extent in the substance of the cerebral tissue subjacent to the affected meninges. The tuberculous process may involve to a slight extent the pia mater of the spinal cord, especially of its upper portion. An obliterating endarteritis and phlebitis may be present.

The degree of pathological change varies with the case. Miliary tubercles may be very numerous, and be found at the convexity as well as at the base; or they may be so few that they easily escape notice. In such cases they can be discovered by careful investigation along the course of the vessels, especially in the anterior and posterior perforated spaces. The membranes at the base may be only slightly turbid, or in long-continued cases much thickened, gelatinous, and matted; the ventricles contain only a normal amount of fluid, or be much distended; the exudate clear or seropurulent, watery or gelatinous; the brain-substance firm or show the superficial congestion and softening of a meningo-encephalitis.

In many instances, particularly in infancy, the lesions of a widespread miliary tuberculosis are observed in addition to that of the meninges. In others there is an accompanying tuberculous bronchopneumonia; or, in older children, tuberculosis of the bones or joints. Involvement of the tracheobronchial glands is found in most cases; less often of other lymphatic glands; or a small, inactive focus in the lungs may be discovered. In most instances the existence of tuberculosis in other regions than the meninges is not discoverable until at autopsy.

Symptoms.—In what may be called the typical form, it has been often customary to divide the symptoms into three stages. To these various titles have been given, very fitting ones being; (1) *the stage of irritation*, or the prodromal stage; (2) *the stage of pressure*, or the transitional stage, this being sometimes called the stage of irritation; (3) *the stage of paralysis*, or of coma. These periods however, are never sharply demarcated, and, in general, the disease is subject to so many variations that it is as well to make no attempt to follow such a classification too exactly.

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[1] Jour. Amer. Med. Assoc., 1912, LIX, 165.

The onset is usually slow, but not infrequently in infants, acute. There develop irritability; fretfulness; malaise; sometimes drowsiness; disinclination to play; perhaps headache, discovered if the children are old enough to complain of it; loss of appetite; constipation; vomiting without discoverable cause; restless sleep with dreaming or grinding of the teeth. Sometimes the mental state and the disposition are strangely altered. The temperature is often slightly and irregularly elevated; sometimes high, but oftener moderate. It is impossible at this time to come to an accurate or even a provisional diagnosis, and the children are supposed to have some one of a large range of maladies other than the true one.

The second stage, that of pressure, then begins with a sudden or gradual increase in the severity of the symptoms, and distinct cerebral manifestations appear. Most marked here is the development of unusual drowsiness, ushered in or not by a convulsion. The child lies still, with the eyes half-open and apparently fixed on some distant object; and when roused with difficulty from this drowsy state cries out or may become delirious. With the stupor are rigidity of the neck and often of much of the body; slowness or irregularity of the pulse; irregular or sighing respiration; and a tendency to assume the "gun-hammer" position; *i.e.* with the head thrown backward, the back arched, the, thighs flexed on the abdomen, and the arms flexed at the elbows and drawn to the sides of the thorax (see p. 330). In other instances the position assumed is on the back with the arms extended along the sides of the trunk, and perhaps with the head turned to one side. There is headache; sometimes photophobia; moderate hyperesthesia; a disposition to sudden screaming, particularly at night; *i.e.* the so-called "hydrencephalic cry;" exaggerated reflexes; scaphoid abdomen; bulging of the fontanelle in infancy; and often repeated general convulsions, or mere twitching of some of the muscles. The eyes frequently are turned to one side or rolled in different directions, or there may be nystagmus;

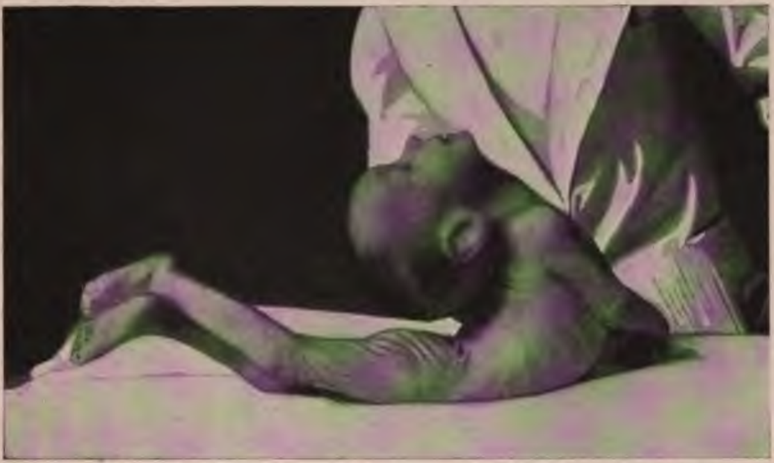

FIG. 344.—TUBERCULOUS MENINGITIS WITH AN UNUSUAL DEGREE OF OPISTHOTONOS.

From a patient, aged 6 months in the Children's Hospital of Philadelphia. Had had opisthotonos for 3 weeks. The occiput was almost against the shoulders, and the heels sometimes touched the head, tubercle bacilli in the spinal fluid.

the pupils are contracted or normal; vomiting may occur at intervals. Fever is present, but not often high and occasionally is absent. During this stage there is a well-marked *tâche cérébrale* with which is frequently associated an irregular flushing of the body, as of the trunk, following the exposure of the surface to the air; or seen as a suffusion of the face, which comes and goes slowly and without discoverable reason for the change.

The symptoms gradually grow worse, and after a variable and uncertain time the evidences of irritation from pressure give way largely to those of the paralysis of the final, or third, stage of the disease. Convulsions and rigidity of the neck, however, often persist. There is now complete coma; the patient rapidly loses strength; the pulse becomes more accelerated and irregular; paralysis is widespread; voluntary movements cease; the reflexes are abolished; the pupils respond little if at all to light and are dilated; the cornea is insensitive to touch; the respiration is irregular, sometimes rapid, and sometimes approaching the Cheyne-Stokes type. The temperature may often rise to 104 or higher, or may be normal or even subnormal; there is incontinence of urine and feces, or the urine may be retained; swallowing is difficult or impossible. The occasional occurrence of glycosuria has been emphasized by Frew and Garrod.[1] Death takes place in coma with or without convulsions.

Certain of the symptoms referred to, and others not mentioned in detail, require a fuller elaboration.

Clonic convulsions, although oftenest seen in the stage of pressure, may occur at any period of the disease; being sometimes initial, sometimes observed just before death. They may be general, or limited to certain localities. They may, for instance, occur in the form of a constant rhythmic twitching of a hemiplegic distribution lasting for hours, and then disappearing or being replaced by convulsive movements in other parts of the body. Not infrequently widespread convulsions do not occur at all; and they are as a rule less frequently seen than in other forms of meningitis, and occur oftenest in the 1st year.

Rigidity of various distribution is a prominent symptom. That of the neck occurs early among the manifestations of pressure. It may persist throughout the attack, although it often is absent for a time and then returns. There is usually not so much retraction of the head as in cerebrospinal fever. One or more limbs may develop a tetanic rigidity. Opisthotonos is usually not so well marked as in cerebrospinal fever, but is sometimes excessive (Fig. 344). Distention of the abdomen may take the place of retraction, especially in infants.

The tendon *reflexes* are increased until coma is well established, when they may be abolished. The spastic condition of the limbs often makes the examination unsatisfactory. Kernig's sign (Fig. 344, p. 322) may or may not be present. I have not found it a sign of any special diagnostic value in tuberculous meningitis. The Babinski reflex (p. 234) is frequently present in children 3 years of age or over. Before this age it is without significance. Brudzinski's reflexes, identical and contralateral (p. 322), are usually observed as in other forms of meningitis.

Ocular symptoms are of interest. The pupils are often contracted in the earlier stages, dilated in the last. Moderate swelling of the disc is frequently seen early; choked discs not so often. Tubercles may be present in the choroid, oftenest in the cases where tuberculous meningitis is but one of the manifestations of general miliary tuberculosis. Paralysis of the movements of the eyeballs is common, either of the oculo-motor or the abducent nerve. It is usually unilateral. As the disease advances the eyes are not shut completely, and the cornea loses its sensitiveness to touch and becomes injected and cloudy.

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[1] Lancet, 1913, I, 15.

Paralysis may be widespread in the parts supplied by other cranial nerves, or may involve other regions of the body. It is often hemiplegic in type, but may be paraplegic or monoplegic, or may change from time to time. On the whole, paralyses are less frequent than convulsive symptoms.

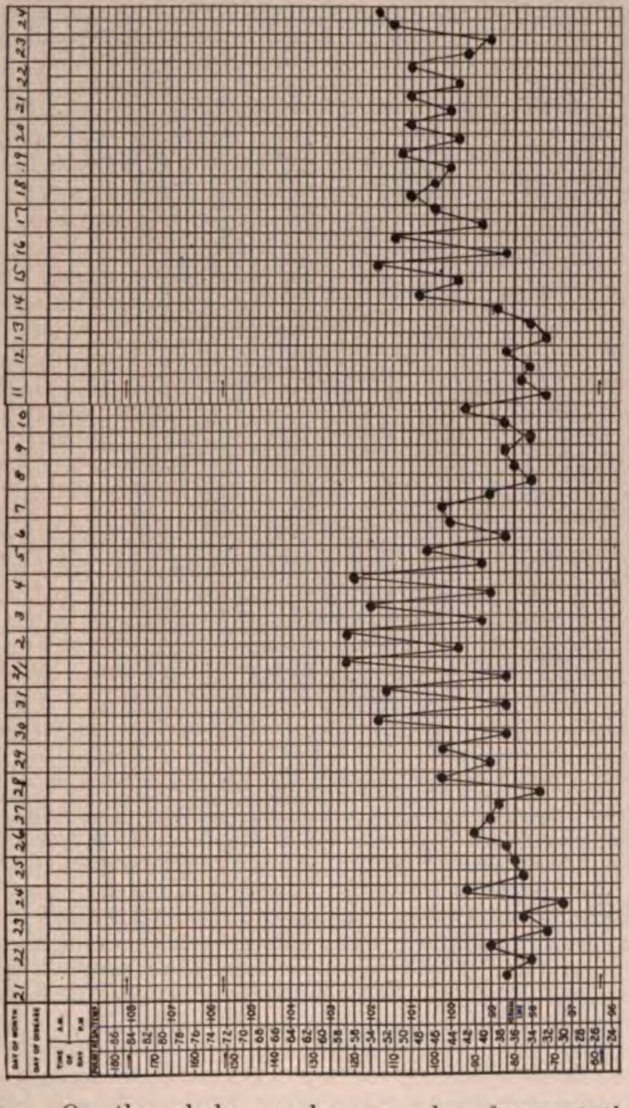

FIG. 345.—TUBERCULOUS MENINGITIS.

James L., aged 6 years. Admitted to the Children's Hospital of Philadelphia, Jan. 1, suffering from hip-joint disease and abscess of the jaw; Feb. 6, vomiting began and continued, and other evidences of meningitis developed; Feb. 15, tubercle bacilli found in the spinal fluid. Date of onset probably with rise of temperature on Jan. 28.

Although a constipated condition of the *bowel movements* is the rule, it is to be remembered that diarrhea is not a contra-indication to the diagnosis of tuberculous meningitis. This latter symptom is found oftener in infants than after this period. Constipation occurs most frequently early in the disease. *Vomiting* is one of the most characteristic of the early symptoms. It is not attended by nausea or by retching. It takes place at irregular intervals, and usually not many times in a day. As the disease advances vomiting sometimes grows more projectile, the contents of the stomach being ejected for a distance of several feet. Generally it is now not so frequent, and may cease entirely as the stage of pressure advances.

The *temperature* is variable and uncharacteristic. It may rise at no time to over 100°F. (37.8°C.), but as a rule there is slight irregular fever throughout the prodromal period, becoming somewhat higher in the stage of pressure, but still tending to be moderate and generally not reaching over 102°F. (38.9°C.); (Fig. 345); while shortly before death the temperature often reaches 104°F. (40°C.) or more (Fig. 346), or sometimes becomes subnormal.

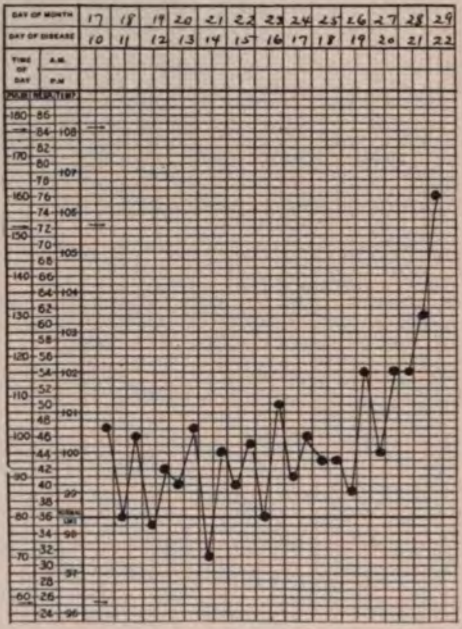

FIG. 346.—TUBERCULOUS MENINGITIS WITH LOW DEGREE OF FEVER UNTIL HYPERPYREXIA ON THE LAST DAY.

Catherine McC., 8 months old. Admitted to the Children's Hospital, Dec. 17. Ten days previously began to vomit, apparently result of bad feeding, was constipated; Dec. 19, examination negative except for rapid cardiac action; Dec. 20, drowsy, abdomen distended, Cheyne-Stokes respiration, neck-reflex marked. Lumbar puncture revealed tubercle bacilli with polymorphonuclear cells 82.7 per cent. and lymphocytes 17.3 per cent.

An early rapidity and irregularity of the *pulse* is of no diagnostic value; but the slowness and irregularity which is a symptom of the fully developed second stage is of great importance when present. This is, however, not infrequently absent in infancy. During the final stage of paralysis the pneumogastric nerve is involved, and the pulse consequently becomes rapid.

While it is true that the *tâche cérébrale*—the broad red line produced by drawing the finger-nail over the skin—is not a pathognomonic sign, occurring as it does in any form of meningitis as well as in other conditions, yet in association with other symptoms it is extremely suggestive (p. 322, Fig. 340). Although seen at its best in the stage of pressure, it may occur well marked among the prodromes. I have seen well developed tâche, discovered by accident and without evidence or suspicion of meningitis, soon followed by characteristic symptoms of the disease.

The *cutaneous tuberculin reaction* of von Pirquet is of limited diagnostic importance. It is usually discoverable in the earlier stages, and is of corroborative value if suspicious symptoms are present; but when the attack is well advanced it may be absent.

The *blood* exhibits often little, if any, increase in the number of white corpuscles early in the course of the disease, but as the attack advances there is generally some degree of leucocytosis, chiefly of the polymorphonuclear cells, equalling about 20,000 or less per c. mm. The leucocytosis is, therefore, less marked, as a rule, than in cerebrospinal fever. Morgan[1] found an average of 20,900 in 252 examinations, only 10 cases being below 10,000. The polymorphonuclear cells averaged 72.6 per cent.

Examination of the *spinal fluid* obtained by lumbar puncture (see p. 235) is of great importance. The fluid generally flows under increased pressure, reaching about 50 mm. of mercury (Pfaundler),[2] and is either limpid, even with a black back-ground, or shows only a faint haziness with minute floating particles. The specific gravity is 1010 to 1011; Noguchi's globulin test is positive, as in all forms of meningitis, and the sugar-content is diminished. When examined microscopically a few cells are obtained, numbering from 100 to 200 or more to the c. mm. There has been much discussion over the nature of the cells found. Although they would appear to be usually nearly all mononuclear, yet it is certain that a predominance of polymorphonuclear cells does not exclude tuberculous meningitis. To discover tubercle bacilli present the last portion of the fluid obtained should be placed in a sterilized test-tube, corked with raw sterilized cotton, and allowed to stand for 12 to 24 hours. Generally a very delicate fibrin coagulum forms by this time, and this is further proof of the inflammatory nature of the exudate. In the coagulum are enmeshed most of the tubercle bacilli. It should be drawn out carefully upon a cover-slip, dried and stained. The bacilli are usually but few in number, and prolonged search may be required to detect them. They may, however, be found in the majority of cases, in my experience. Bacilli may not be obtained at the first puncture, but become discoverable at later ones as the disease progresses. If no coagulum forms, the fluid should be centrifugated, and successive drops, taken from the bottom of the centrifugating tube, dropped upon a cover-slip, evaporated, stained, and examined. This concentration of the fluid may render the search easier.

Course and Duration.—The total duration of the disease is variable. Generally it is from 2 to 3 weeks after definite symptoms appear; sometimes 4 weeks or even longer in all; and sometimes apparently very short, lasting only a week or less. The prodromal symptoms last a variable time with but little change, averaging perhaps about a week or a little more, but varying from a few days to 2 or 3 weeks. The duration of the second stage can hardly be computed, as it begins and ends in so indefinite a manner. An average of a week would probably cover this also. From the time the stage of coma fully sets in, life is generally of but short duration; usually under or slightly over a week; but not infrequently the terminal stage is prolonged remarkably, the child living day after day in a condition in which it seems that death is to be expected at any moment. Death takes place usually quietly in coma. Much less often there are terminal convulsions.

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[1] Amer. Journ. Dis. Child., 1916, XI, 224.

[2] Jahrb. f. Kinderheilk., 1899, XLIX, 264.

The course is subject to great variations. It sometimes happens that the prodromal stage is absent, or so slightly marked that it has been overlooked by the parents. This is especially true of infants in the 1st year. Perhaps careful interrogation will show that the child has not been quite well during this period. In other cases the onset of symptoms is actually sudden and the course very short. I recall one child under hospital observation for moderate rickets, who was about to be sent home. Convulsions suddenly developed, followed by death in less than 24 hours; and autopsy showed tuberculous meningitis. In other instances a unilateral convulsion is followed by hemiplegia, or there may be some other initial localizing symptom, which may in 1 or 2 days give place to the ordinary prodromal symptoms of tuberculous meningitis. In others prodromal symptoms have been present for some time, and the disease then terminates in convulsions and death; the ordinary stages of pressure and paralysis being almost absent. Still other cases exhibit the usual symptoms of the disease throughout, but with greater intensity and of but short duration, the attack terminating fatally in a few days; while others, as stated, reach the final stage, and then live day after day in a condition where death seems momentarily imminent. Exceptionally the attack may begin with a local paralysis of one of the cranial nerves, oftenest the abducent; or may be characterized by psychic symptoms of a violent nature.

Temporary improvement for some hours, or even for as long as 2 weeks, may occur in the stage of pressure; the stuporous state passing and the child even awakening bright and ready to play; but the amelioration is not a lasting one and the serious symptoms again appear. The occurrence of this improvement is especially true of patients past the period of infancy. Even a remission for months may be witnessed. It is a question whether or not these instances of long remission should be considered as recovery followed by recurrence. As a general rule the course in infancy is more abrupt in its onset; general convulsions are more frequent; the abdomen is less often retracted; the symptoms are less characteristic; diarrhea is oftener seen than in older subjects; and the course is shorter.

Prognosis.—The prognosis is practically absolutely unfavorable. A number of cases of reported recovery are on record. Pitfield[1] collected 29 cases including one of his own; and Bókai[2] 27 cases in which the presence of tubercle bacilli was proven by examination of the spinal fluid or by animal inoculation. The two lists do not duplicate each other completely; and a few other reported instances could be added. Any case to be placed properly in the list of recoveries should be watched for months to make sure that it is not an instance of remission; should exhibit tubercle bacilli in the cerebrospinal fluid, and should have it made absolutely certain by cultural and inoculation tests that the organism found is not by any chance an acid-fast bacillus other than that of tuberculosis. That even long remissions may occur lasting perhaps several months is undoubtedly true; and a number of reported cases have been collected by Martin.[1]

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[1] Amer. Journ. Med. Sci., 1913, CXLVI, 37.

[2] Jahrb. f. Kinderheilk., 1914, LXXX, 133.

Diagnosis.—This is not difficult in well-marked cases, but often far from easy in the early stages. There is little at the beginning to awaken suspicion, and that of the existence of some *digestive disturbance* is generally entertained. As the prodromal stage advances without relief by treatment, the possibility of tuberculous meningitis should be thought of, but the diagnosis should be very reserved. The most suggestive symptoms are vomiting without evidence of indigestion; constipation; drowsiness, and a well-marked tâche cérébrale. As the later stages are reached, the diagnostic symptoms of importance are slowness and decided irregularity of the pulse; stupor; convulsive twitchings; paralysis; irregular respiration; and rigidity, especially of the neck. In all doubtful cases a lumbar puncture should be made. The presence of MacEwen's sign, viz. a percussion-note over the cranium clearer than normal, is of some value in recognizing the presence of increased intracranial pressure, and is common in tuberculous meningitis, but does not differentiate it from other intracranial diseases in which the tension is above normal.

Tuberculous meningitis is to be distinguished from *typhoid fever*. Generally there is no likeness, but sometimes the resemblance is close. Both diseases have similar mental symptoms in the early stage, enlargement of the spleen, and absence of marked leucocytosis; while rigidity of the neck may be present in each, especially if the typhoid fever is accompanied by meningismus or serous meningitis. The presence of diarrhea, roseola, and the Widal reaction will prove the existence of typhoid fever; while the results of lumbar puncture will establish that of tuberculous meningitis.

Pneumonia does not often cause any diagnostic difficulty. It frequently begins, it is true, with symptoms pointing to meningitis; but these suggest some other form rather than that dependent upon tuberculosis. From *poliomyelitis* the diagnosis is in some cases at first difficult or impossible. This applies especially to the forms of this disease in which meningeal symptoms are well marked. The cerebrospinal fluid exhibits almost an identical condition in both, except in the matter of the tubercle bacilli. The onset of poliomyelitis is usually more sudden; that of tuberculous meningitis insidious. Yet often only the passing of time will make the diagnosis manifest, the meningitic symptoms of poliomyelitis disappearing and paralysis developing.

Tuberculous meningitis is to be differentiated from *other forms of meningitis*. In general the longer continuance and the lower grade of fever characterize tuberculous meningitis. The *purulent forms*, as represented by the acute stage of cerebrospinal fever and by cases due to pyogenic and other germs, are of much more rapid and severe onset; there is higher fever; higher leucocytosis; and the cerebrospinal fluid obtained by puncture is more purulent and contains chiefly polymorphonuclear leucocytes.

Chronic basilar meningitis of infancy has fluid which is generally clear without the presence of organisms, and with a few mononuclear cells; resembling in this last the condition characteristic of tuberculous meningitis. The course is, however, chronic; the disease is a sequel, at least in most cases, to an acute attack of cerebrospinal fever (see p. 336); there is no paralysis or fever, and there is very decided opisthotonos.

Serous meningitis sometimes occasions the greatest difficulty in diagnosis. The spinal fluid in this disease is clear, without microörganisms, and exhibits a few mononuclear cells as in tuberculous meningitis. In the latter, however, there is usually a somewhat greater number of cells, and the onset is slower, with characteristic symptoms. Only continued study and the search for tubercle bacilli, perhaps with the inoculation of a guinea-pig, may solve the question in some instances. The more favorable outcome of serous meningitis frequently makes the final diagnosis certain. I have seen cases of serous meningitis finally recover, in which there had been every reason from a clinical point of view to consider the disease tuberculosis, except that no tubercle bacilli were found.

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[1] Brain, 1909, XXXII, 216.

Treatment.—Practically the only treatment open to us is that of *prevention*. Allowing little children to live in a house where tuberculous adults are or have recently been present is a very dangerous procedure. The infants, ordinarily on the floor, easily soil their hands and then carry the germs to their mouths. Children who have had measles or pertussis should have their convalesence carefully favored in every possible way, as by change of locality if necessary. The general health among children is to be kept in the best possible condition, particularly if there is any recent tuberculous ancestry, or if other children in the family have suffered from tuberculosis.

As far as medical knowledge extends, the therapeutics of tuberculous meningitis actually developed is unavailing. It is useless to try the various ointments, blisters and other applications, hot baths, ice-caps, blood-letting, and the like, which have been recommended from time to time. Repeated lumbar puncture sometimes gives temporary relief, and may well be employed on the ground that the diagnosis may be a mistaken one; or that the case may turn out to be one of the rare instances of recovery. It may be combined with the intraspinal injection of hexamethylenamine. For the same reason every effort should be made to sustain the patient's strength. Direct draining of the ventricle has been tried, but without good results, and I have seen no success with draining of the cisterna magna. The procedure could avail only by relieving intracranial pressure; and it would not appear that this is the direct cause of death. Moreover, tuberculous meningitis is often only the terminal manifestation of a more widespread tuberculosis, which relief of the intracranial symptoms could in no way benefit.

CHRONIC MENINGITIS

In the majority of instances this occurs as a final stage in many cases of cerebrospinal fever. This form, eventuating in a chronic hydrocephalus with progressive wasting, has been described in connection with that disease. In addition there is a form of chronic meningitis, now under our consideration, which occurs especially in infants and is known under the name of *chronic basilar meningitis* or *posterior basic meningitis*. It was described by Gee and Barlow[1] and later by Still[2] and others. It probably represents a sporadic infantile form of cerebrospinal fever, although this is not universally admitted.

Etiology.—The large majority of these cases are seen in the 1st year of life, and after the 2d year it is rare. The cause appears to be a diplococcus which resembles and probably is identical with that found in ordinary cases of cerebrospinal fever, although, according to Houston and Rankin[3] the opsonic and agglutinative reactions are different.

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[1] St. Barthol. Hosp. Rep., 1878, XIV, 23.
[2] Journ. Path. and Bact., 1898, V, 147.
[3] Brit. Med. Journ., 1907, II, 1414.

Pathological Anatomy.—The lesions are generally found only at the base of the brain and upper part of the cord, and consist of thickening of the pia mater with the production of firm adhesions, obliteration of the foramina, and consequent accumulation of fluid followed by compression of the cranial nerves and more or less distention of the ventricles.

Symptoms.—After a gradual onset, or after a mild attack of cerebrospinal fever, there develop muscular rigidity of the limbs, retraction of the head, and sometimes finally a remarkable degree of opisthotonos. There is wasting; exaggeration of the reflexes; often blindness, yet without optic neuritis; irritability; and headache or dullness; but coma does not often occur unless as a terminal symptom. There are also nystagmus; vomiting; and an irregular fever or none at all except at the beginning, or there may be afebrile periods alternating with others of high temperature of short duration. The fontanelles bulge, showing the presence of moderate hydrocephalus. The arms are flexed at the elbows; the hands clenched; the legs and feet usually extended and adducted. Convulsions may occur, but less frequently than in acute meningitis. These symptoms vary from time to time in the same case. Lumbar puncture gives a turbid fluid in the early stages, but later this is clear or none at all is obtained.

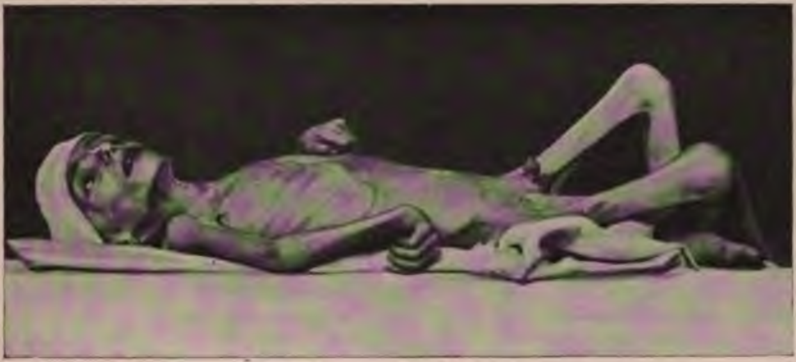

FIG. 347.—CHRONIC MENINGITIS.

Child of 4 years, a patient in the Children's Hospital of Philadelphia. Duration of disease uncertain. Acutely ill 6 weeks before admission, with persistent wasting and often unconsciousness. Temperature afebrile while in the hospital. Examination of spinal fluid negative. Gradually lost ground and died 4 months after admission.

Course and Prognosis.—The course of the disease is slow, the attack lasting for several months, and sometimes even for over a year; the child meanwhile growing constantly weaker and more emaciated (Fig. 347). The prognosis is very unfavorable, nearly all the cases ending fatally. Death is usually the result of the progressive loss of strength, but may be brought about by convulsions or some intercurrent disorder; or may occur suddenly without discoverable reason. In the few cases which recover—generally only in subjects past the age of infancy—the convalescence may be complete or a hydrocephalus may remain.

Treatment.—Early in the attack serum should be employed as in any form of meningococcic meningitis. After this period treatment is unsuccessful. Repeated lumbar puncture should be done in the effort to prevent excessive distention of the ventricles.

Chronic Syphilitic Meningitis.—A chronic leptomeningitis is occasionally seen the result of hereditary syphilis. The lesions vary in position, and the symptoms correspondingly. The characteristic pathological changes consist in thickening, opacity, and adhesion of the pia-arachnoid over the vertex or the base, with some shrinking of the convolutions, and often hydrocephalus. When the base of the brain is involved the clinical manifestations may be similar to those seen in the chronic basilar meningitis just described. When the vertex is especially affected the symptoms are of a different nature, and, as seen in infancy, consist principally in lack of intellectual development; inability to hold up the head; and diminished power in the extremities, perhaps with rigidity. A great variation in the symptoms may, however, be observed. The diagnosis rests upon the presence of other manifestations of syphilis, or the obtaining of a Wassermann reaction. The prognosis is unfavorable, the employment of mercury, the iodides, or of arsphenamene being generally useless except in the early stages, before the condition has become chronic and the diagnosis has been made.

CHAPTER V

DISEASES OF THE BRAIN-SUBSTANCE AND VESSELS

CEREBRAL HYPEREMIA

This symptom may be either of an *active* or a *passive* nature. The former may be the result of over-eating; exposure to the sun; the use of alcohol; undue mental work or excitement, and possibly the existence of the acute infectious diseases and disorders of the kidneys. There occurs an over-filling of the brain with arterial blood. The condition plays a very slight rôle in the production of symptoms in early life.

Passive hyperemia, with a congestion of the brain with venous blood, may be seen in chronic diseases of the heart with dilatation of the right side of the organ. It may occur, too, in any disorder which interferes with respiration and induces distention of the right cardiac chambers. Here may be mentioned general convulsions; laryngospasm; pseudomembranous laryngitis; straining at stool; severe coughing, and the like. Pressure of any growth upon the jugular vein or the descending cava constitutes other cause.

The **symptoms** of cerebral hyperemia may be of a temporary or of a more persistent character, according to the case. They consist in distention of the veins of the head and neck; vertigo; headache; sleeplessness; and sparks before the eyes. The condition may be so severe that convulsions occur; or stupor or, finally, coma may develop. The prognosis depends upon the nature of the case, and the **treatment** is directed chiefly to this. In addition cold compresses applied to the head may be of benefit in some instances; and in others the keeping of the head in an elevated position is of service.

CEREBRAL ANEMIA

Etiology.—The causes of this condition are various, among them being sudden hemorrhage or the development of general anemia produced in other ways, and the presence of passive hyperemia or of cerebral edema; either of which may be finally attended by compression of the small vessels of the brain and consequent diminished supply of blood to the organ. In addition, the occurrence of sudden fright or other shock, the sight of blood, etc., may produce a sudden anemia by weakening of the heart's action, or through other vasomotor disturbance.

Symptoms.—These may be of an acute paroxysmal or of a more chronic nature. In the former there is ringing in the ears; bright or dark spots before the eyes; dizziness; drowsinesss; yawning; pallor; sweating; nausea; rapid respiration and pulse; and often loss of consciousness—the symptoms, in fact, of syncope. Convulsive movements may sometimes develop. In the more chronic cases there is a condition of mental exhaustion with inability for mental work; noises in the ears; flashing of light before the eyes; great excitability from slight causes; headache; insomnia; and in severe cases delirious states or coma. In marantic infants, especially after prolonged diarrhea, the symptoms of meningismus (p. 316) or of the hydrocephaloid disease of Marshall Hall[1] may develop. There are then observed restlessness; jactitation; rolling or crossing of the eyes; retraction of the head; depression of the fontanelle; feeble and rapid pulse; rapid and often irregular respiration; narrowed and, later, dilated pupils; convulsions, and finally coma. The condition depends probably upon the increasing weakness of the cardiac action, the result of the general inanition. In some cases, however, the disturbance may be an acidosis.

Prognosis and Diagnosis.—The prognosis in the acute cases is generally favorable if the cause can be removed. The diagnosis is sometimes one of difficulty. In the acute severer cases the syncope may suggest the existence of epilepsy, especially if convulsive movements occur; and in the absence of such movements petit mal may be suspected. The presence of some cause for anemia, the longer duration of the attack, and the absence of repetition of this serve to eliminate epilepsy. In the more chronic forms the evidences of anemia elsewhere in the body render the diagnosis easy.

Treatment.—This is primarily that of the cause, but in addition measures must be taken to relieve urgent symptoms during an attack. The patient should be placed in a horizontal position or with the head lowered, the temperature of the body maintained, and the circulation favored by the use of friction of the extremities and the application of heat. Internally, aromatic spirits of ammonia, or whiskey or other liquor may be given. In the worst cases hypodermoclysis or transfusion may be required.

EDEMA OF THE BRAIN

In this disorder there is a saturation of the brain substance with fluid transfused from the vessels. It may be general as the sequel of an agonal process, or be a localized condition dependent oftenest upon some focal disease, such as abscess; tumor; hemorrhage; or embolism or thrombosis of the vessels. It may occur, too, as a result of trauma or of a serous meningitis or may be caused by the passive hyperemia of cardiac disease or by nephritis. The symptoms may appear very rapidly, or after some days of illness. They are to an extent those produced by the original focal lesion; but, as the edema increases, drowsiness and finally coma develop. The diagnosis, especially from cerebral anemia, cannot be made with certainty during life. The prognosis and treatment are those of the original cause.

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[1] Diseases of the Nervous System, 1841, 5, 153. See also Medical Essays, 1825.

EMBOLISM AND THROMBOSIS OF THE CEREBRAL VESSELS

Embolism and thrombosis of the cerebral vessels, with the exception of those of the sinuses, not included here, are of much less frequent occurrence in early life than in the adult.

EMBOLISM

Etiology.—This is oftenest the result of endocarditis, but the various acute infectious diseases are also to be numbered among the causes. The plugging of the arteries may be by a vegetation whipped off from a cardiac valve, or by a portion of a thrombus from the cavity of the heart or elsewhere carried away in a similar manner. The disorder is not common at any time of life, and is oftener seen after the age of 10 years. I have records of 2 instances in children of 11 and 13 years, respectively, suffering from endocarditis.

Pathological Anatomy.—Much most frequently embolism occurs somewhere in the course of the middle cerebral artery, one or more branches being affected, oftener in the cortex than in a central region. The first alteration following the plugging is anemia in the tissues supplied by the vessel. The later changes depend upon the size and position of the embolism and the duration of life. The area involved is softer than normal and at first red from hyperemia and the presence of small hemorrhages, while later the color becomes yellow. Inflammatory changes occur about the softened area; and if the embolism is of a septic nature, suppuration may follow. The final condition is localized sclerosis or the formation of a cyst.

Symptoms and Course.—The onset is sudden, often with convulsions; followed by headache, vomiting, delirium, or coma. These symptoms may continue during several days, with the development of moderate fever and weak, rapid pulse, and may go on to a fatal ending or may disappear. With these general symptoms are the focal ones, the nature and distribution of these depending upon the position of the embolism. The focal symptoms will be referred to again in the section on Cerebral Paralysis (p. 365). The **prognosis** is serious, more than one-half of the cases dying in coma with increasing cardiac weakness. Those which recover exhibit permanent localized sequels, unless the original lesion is very small.

Diagnosis.—This must be based chiefly upon the sudden onset, combined with the existence of chronic disorders known to favor the production of embolism. The disease is especially liable to be confounded with *cerebral hemorrhage*. The chief distinction is the more rapid disappearance of the general cerebral symptoms in the cases of embolism which recover. Cerebral *thrombosis* is slower in its onset; otherwise the symptoms are much the same. Acute *hemorrhagic encephalitis* may be distinguished with difficulty, except for its greater early elevation of temperature and the less rapid onset of paralytic symptoms.

Treatment.—The treatment is entirely symptomatic in the early stage of the disease; the effort being made to strengthen the weak cardiac action by stimulants, and to relieve symptoms of nervous excitation. The management of the paralytic manifestations remaining will be discussed elsewhere. (See Cerebral Paralysis, p. 372.)

THROMBOSIS

Thrombosis of the cerebral arteries or veins is uncommon in early life. The latter suffer much more frequently than the former. It is nearly always the result of an endarteritis or endophlebetis dependent upon hereditary syphilis. It may also occasionally be produced by extreme feebleness of the circulation, or of the extention of the process from a sinus thrombosis. The disease occurs much less frequently after the age of 10 years. The presence of a positive Wassermann reaction and of other evidences of syphilis may often be discovered.

The pathological processes which develop after the thrombosis has formed are identical with those seen in embolism. The **symptoms** are slower in onset than in that condition; in general less striking; and the production of paralysis is slower, requiring sometimes even several days to develop fully. Defective intelligence is a frequent sequel. In other respects the symptoms are the same as attend embolism. The prognosis is unfavorable for complete recovery. There is also the danger of recurrence, although there is a good hope of the prevention of this by appropriate treatment. The diagnosis rests especially upon the slowness of onset and the history of former, or the discovery of present, manifestations of syphilis. The **treatment** consists at first in that suitable for embolism; but very promptly systematic antisyphilitic medication should also be commenced, especially by mercurial inunction, and later by the administration of the iodides.

SINUS-THROMBOSIS

(Thrombosis of the Cerebral Sinuses)

Etiology.—Thrombosis of this region occurs in early life more frequently than that of the cerebral veins or arteries, but is still not a common affection. The disease falls naturally into two classes based upon the etiology and pathology: the first the marantic or primary thrombosis; the second the inflammatory or secondary form. As the name implies, *marantic sinus-thrombosis* occurs in connection with greatly debilitated states accompanied by feebleness of circulation or alteration of the character of the blood, such as may be seen in the course of severe, long-continued gastrointestinal disease, prolonged suppurating processes, tuberculosis, syphilis, and the acute infectious diseases. Sometimes the shock of an operation will be followed by a thrombosis of the sinuses. The more frequent *inflammatory* form is an extension of inflammation from neighboring regions, such as disease of the ear, orbit, nose, or bones of the cranium; traumatism; inflammatory processes within the skull; suppuration situated on the face or scalp; facial erysipelas; and septic conditions in the mouth or pharynx, as in diphtheria or scarlet fever. Of the causes in this form the most frequent in early life is inflammation of the ear.

Bacteria of various sorts play an important rôle in the production of sinus-thrombosis. In cases of inflammatory nature germs of some sort are always present, as in those, too, of the marantic form which depend upon suppurative processes in distant parts of the body; and there is a tendency for every thrombosis, however produced, finally to exhibit bacteria and consequently to be septic. Among the various germs which have been found streptococci appear to be those most frequently present. Marantic thrombosis occurs oftenest in early childhood, and especially in infancy. Inflammatory cases may develop at any age, but appear less frequent in the first 10 years of life than in the period from 10 to 30 years, so far at least as that of otitic origin, the most common inflammatory form, is concerned (Hessler).[1]

Pathological Anatomy.—In the *marantic* cases the wall of the sinus is entirely unaffected and there is found only a fairly firm clot, more or less adherent to the vessel, and of a color varying from dark-red to a more yellowish-red tint depending upon the age of the lesion. The clot fills the sinus, causing it to stand out as a firm hard cord. The veins emptying into it exhibit a similarly corded appearance. The surface of the brain in the vicinity of the thrombus may sometimes be deeply congested, and sometimes covered with effused blood. The meninges may be congested or inflamed, and in some cases there is effusion of blood into the ventricles. The most frequent seats of marantic thrombosis are the superior longitudinal sinus and the lateral sinuses, and the process may extend secondarily into most of the sinuses of the brain.

In the *inflammatory* variety there is a phlebitis present in addition to the thrombus, the wall of the sinus exhibiting evidence of inflammation. The clot in this form is discolored, and may show purulent softening. The meninges may be locally or generally inflamed, and the brain-substance exhibits congestion or hemorrhage, perhaps with areas of softening or even abscess. Pyemic abscesses may be discovered also in other organs of the body.

The seat of the thrombosis in the inflammatory form depends upon the location of the primary process. As this is in the ear in most instances, the thrombosis is most frequently in the lateral sinus. Involvement of the cavernous sinus is due oftenest to disease of the nasal and accessory cavities, but sometimes to that of the orbit, the mouth, or the base of the skull; or it may occur secondarily to thrombosis of the lateral sinus. Thrombosis of the petrosal sinus is seen generally only in combination with that of the lateral or cavernous sinus; that of the longitudinal sinus, when of an inflammatory nature, follows disease of the vault of the cranium or of the scalp covering it.

Symptoms.—The clinical evidences of any variety of sinus-thrombosis are uncertain and obscure. This is especially true of the *marantic* form, in which the symptoms, if any, are overshadowed by those of the primary disease. Prodromes may be present, such as vomiting and headache; followed promptly by the development of delirium, hyperesthesia, rigidity, convulsions, paralysis, somnolence, or coma. Fever is generally high, but sometimes absent. In some cases there are the external evidences of interference with the circulation, as severe nose-bleed and dilatation of the veins of the scalp and face. If it is the lateral sinus which is involved, the jugular upon the side affected may be less full than normal, and there may be cyanosis and edema upon one side of the head. As a rule, however, the marantic cases run a latent course and are unrecognized during life.

The symptoms of *inflammatory* sinus-thrombosis are more characteristic, and the evidences of decided sepsis are generally present. Among the symptoms are headache; irregular and extreme rises and falls of temperature with chills and sweats; irregular, weak and rapid pulse; pallor; somnolence, coma, or delirium with jactitation; convulsions; leucocytosis; and perhaps signs of pyemic abscesses in other parts of the body, with the discovery of pyogenic germs in the blood. In addition there are various local manifestations depending upon the sinus which is involved. Thrombosis of the lateral sinus secondary to otitic disease produces dilatation of the superficial veins, with a swelling and tenderness in the mastoid region partially dependent upon edema. Sometimes the jugular vein is involved in the process, becoming tender, cord-like, and hard, and the head may be bent toward the affected side. Thrombosis of the longitudinal sinus gives rise to fullness of the veins and edema of the forehead, temples, and top of the head; cyanosis of the face; nose-bleed, and papillitis. Thrombosis of the cavernous sinus occasions edema of the eye-lids, usually on one side, and of the root of the nose; exophthalmos; dilatation of the veins of the forehead; paralysis of the ocular muscles, and sometimes papillitis or optic neuritis.

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[1] Die otogene Pyämie, 1896, 438.

Course and Prognosis.—The prognosis of sinus thrombosis is unfavorable. The marantic cases last generally only a few days; sometimes several weeks; pulmonary embolism being a frequent complicating cause of death. In the inflammatory cases death takes place in from a few days up to 3 weeks through pyemia, meningitis, or encephalitis; the prognosis being entirely unfavorable except in those instances where the position of the disease makes operative interference possible. This is chiefly true of involvement of the lateral sinus.

Diagnosis.—The diagnosis of marantic thrombosis is generally impossible during life. It rests principally upon the existence of a primary disease of a nature which is liable to be followed by this condition, and upon the engrafting on the symptoms already present of those pointing to an intracranial affection. Yet meningitis, cerebral paralysis, and encephalitis may produce symptoms similar to those of thrombosis. The diagnosis of the inflammatory form varies with the primary lesion. The principle symptoms have already been sufficiently dwelt upon. Characteristic of each variety is the brown-red or green-red discoloration of the cerebrospinal fluid obtained by lumbar puncture (Finkelstein).[1]

Treatment.—That of the marantic form is of little value, inasmuch as the primary disease had already advanced in most cases to the beginning of a fatal ending. The treatment of the inflammatory form is surgical, and this is applicable especially to cases of thrombosis of the lateral sinus. Prompt operation in the course of otitic disease where the beginning of thrombosis is feared, has been successful in a large number of cases. Körner[2] reports 180 recoveries in 308 cases. The internal jugular on the affected side may be tied or excised and the lateral sinus opened.

INTRACEREBRAL HEMORRHAGE

Hemorrhage connected with the meninges has already been described (p. 314). Here only is to be considered the effusion of blood from the vessels into the brain-substance itself. While meningeal hemorrhage is of common occurrence and the most frequent source of cerebral paralysis, intracerebral hemorrhage is distinctly uncommon in early life and particularly in infancy.

Etiology.—The most frequent cause is trauma. Too great pressure by forceps, the occurrence of severe asphyxia directly after birth, and especially prolonged compression of the head during parturition, may each act as a cause by producing rupture of the vessels. Infectious diseases, including sepsis and preëminently pertussis, are occasional causes, the accident in the last-mentioned being chiefly the result of the increased blood-pressure occasioned by a paroxysm of coughing. Possibly diseases

[1] Charité Annalen, 1898, XXIII, 405.

[2] Die otitische Erkrankungen des Hirns, 1903, 3 Aufl., 118.

of the heart or kidneys may, in rare instances, increase pressure and produce hemorrhage in early life. The endarteritis of hereditary syphilis is among the causes, as are leukemia and the diseases of the hemorrhagic diathesis. Intracerebral hemorrhage may also arise in connection with embolism and thrombosis of the vessels, sinus-thrombosis, encephalitis, and tumors. It seems very probable that convulsions in some instances give rise to hemorrhage instead of being the result of this.

Pathological Anatomy.—The bleeding may be either arterial or venous; oftenest the former. There occurs a rupture of one or more small arteries, generally branches of the middle cerebral artery; or of the veins, most frequently those emptying into the longitudinal sinus. This is the result of degenerative changes in the vessel-walls; except in the case of trauma, where entirely healthy vessels may be injured. Only occasionally in early life does the pathological alteration consist in the production and rupture of a miliary aneurism. The hemorrhage may vary in amount from one of massive size to that of a small pin-head. When it is large the surrounding brain-substance is torn and commonly exhibits punctiform hemorrhages, softening, and edema. The smallest capillary hemorrhages may be entirely absorbed without leaving any discoverable change. In those of larger dimensions there occurs later an encapsulation of the effused blood, either with final absorption and the replacement of the damaged brain-substance by sclerotic tissue, or with the formation of a cyst, or of an area of encephalitic softening with the development of pus.

Symptoms.—The symptoms of intracerebral hemorrhage entirely resemble at the onset those of embolism. There is a sudden loss of consciousness developing with or immediately following the hemorrhage, or occasionally not until several days after the injury. Entire relaxation of the body occurs, with abolition of the reflexes. General convulsions may be seen in some instances. The duration and completeness of the unconsciousness vary with the case, lasting from a few minutes up to several days, coma perhaps continuing until death occurs. In the more favorable cases, as these early general symptoms diminish focal manifestations develop, the general paralytic condition disappearing and the evidences of paralysis being confined to certain regions only. Any convulsions, too, which now occur are limited to the affected regions. As a rule in arterial hemorrhage the focal symptoms are of a hemiplegic type, and as improvement takes place may perhaps become monoplegic only. Venous hemorrhage, occurring as it does oftenest near the sagittal suture and as a result of trauma during birth, is more liable, after the initial coma and convulsions, to exhibit paralysis of a paraplegic type. The subject of post-hemorrhagic paralysis is described more fully elsewhere. (See Infantile Cerebral Paralysis, p. 365.)

Course and Prognosis.—The prognosis is always serious; first of all as regards the danger of death shortly after the hemorrhage, and should this not occur, then with respect to final recovery. Minute arterial hemorrhages may disappear without leaving any symptoms. Very large effusions are followed by death in a few days.

Diagnosis.—This rests upon the sudden onset with the symptoms described, combined with the existence of conditions which are liable to have hemorrhage follow, especially pertussis and trauma. Arterial hemorrhage is distinguished from that of the venous form chiefly by its tendency to develop symptoms of a hemiplegic nature, and by the fact that venous hemorrhage is limited practically to cases occurring at birth. *Embolism* is the disorder most liable to be confounded with hemorrhage, the distinction resting chiefly upon the more rapid disappearance of the general cerebral symptoms in favorable cases of embolism. Intracerebral hemorrhage can be distinguished from *meningeal* hemorrhage only with difficulty, if at all. Focal symptoms are more common in the former; those of irritation in the latter; but the exceptions to this rule are so numerous that it is of little diagnostic value. The very much greater frequency of meningeal hemorrhage in early life, and especially in infancy, makes the diagnosis of this condition always more probable.

Treatment.—Preventive measures are applicable only during and immediately after parturition, and consist in curtailing a too prolonged delivery, and in relieving severe asphyxia as quickly as possible. In the acute stage of hemorrhage the treatment consists chiefly in rest and in sustaining the patient's strength. Early operative interference, with the removal of the clot, could be employed as in meningeal hemorrhage, if the diagnosis can be made of a lesion within reach of surgical intervention. The treatment of the later symptoms developing is considered under Infantile Cerebral Paralysis (p. 372).

NON-SUPPURATIVE ENCEPHALITIS

The form of acute encephalitis described by Strümpell[1] is now very commonly admitted to be but a variety of poliomyelitis (Vol. I, p. 530). There remain to be considered cases of encephalitis depending upon other causes.

Etiology.—The disease may follow any of the acute exanthemata, pneumonia, pertussis, mumps, sepsis, diphtheria, grippe and otitis. Among other causes are trauma, tuberculosis, and gastrointestinal autointoxication. Severe meningitis readily produces a degree of superficial secondary encephalitis (meningo-encephalitis). A form has been described by Virchow[2] as occurring congenitally; and other cases are characteristic of early infancy, and then seem sometimes to be primary. The disease produced in any way is more frequently seen in early life than later, but cannot be called a common disorder.

Pathological Anatomy.—In the congenital form there are small, scattered foci containing round cells and other cells filled with fatty granules, and showing evidences of hyperplasia of the glia. In early infancy the process is generally a *diffuse encephalitis*, and exhibits widespread softening with or without hemorrhagic effusion. The lesions are of a necrotic nature with disappearance of the ganglion cells and atrophy of the white-matter, perhaps followed later by a sclerosing process.

In older children the process is usually more circumscribed, and is oftenest that of *acute hemorrhagic encephalitis*. Any part of the brain may be affected, and usually more than one, although the involvement of the hemispheres generally predominates. There are one or more foci of softening of large or small size with effusion of blood, combined with capillary hemorrhage and cellular infiltration into the surrounding tissue. Bacteria of various sorts may be present. Later a focus may become cystic or sclerosed, or develop into an abscess. The changes are identical with those seen in the polioencephalitic type of poliomyelitis.

A *multiple disseminated myelo-encephalitis* also occurs, not dependent upon poliomyelitis, in which numerous scattered foci appear in both the brain and the spinal cord. Still another form has been described in which there is a *hyperplastic encephalitis* of the hemispheres affecting especially the glia. This is considered under Hypertrophy of the Brain (p. 363).

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[1] Jahrb. f. Kinderh., 1885, XXII, 173.

[2] Virchow's Archiv, 1867, XXXVIII, 129; 1868, XLIV, 472.

Symptoms.—These are variable and uncharacteristic; their nature depending largely upon the position of the lesion and the age of the patient. In the most serious, the diffuse form, as seen oftenest in infants under 1 year of age, the lesions are widespread and the symptoms show themselves in typical cases by a rapid development of high fever, with unconsciousness, convulsions, and coma. The pulse is weak and rapid; there is rigidity of the neck and extremities, and there may be asphyxia and irregular respiration. Focal symptoms may be absent, or only slightly marked. The course is unfavorable, death taking place in 2 weeks or less. Not all cases, however, are so typical as this; since the lesions may be more circumscribed even in early infancy, and the disease milder and followed by recovery.

In the form of the disease with more circumscribed lesions, the more usual variety in children past the age of infancy, there are in severe cases sudden onset, high fever, rigidity, convulsions, vomiting, delirium or coma. Optic neuritis is not uncommon. This condition usually continues but a very short time before distinct evidences of localization show themselves. These indications are naturally of great variety. In the more frequent hemiplegic cases there may be a rolling of the eyes always in one direction; convulsions limited to one-half of the body or to one limb and perhaps Jacksonian in type; paralysis of a hemiplegic form, or limited to one extremity or to the face; tremor on one side of the body only, or a localized diminution of sensibility. I have observed[1] 1 very interesting instance of unilateral tremor in a child of 18 months, which should probably be included in this category. These symptoms last for about a week or more with little alteration, except that after 2 or 3 days the fever generally disappears and increase of tendon-reflexes develops on one side. After this period, in cases which recover, gradual improvement begins and continues during months; usually leaving, however, some degree of paralysis or other evidence of cerebral disorder. In many instances the symptoms described affect both sides of the body.

Many cases are not so severe as the type described. The fever is less high and the general cerebral symptoms less marked as compared with the paralytic manifestations. Sometimes the course of the disease exhibits a more chronic nature, the onset being insidious, and the development of symptoms slow and suggesting those of tumor; yet the patient finally recovering entirely, or with some evidences of cerebral disorder persisting.

The features of encephalitis as described are those for the most part characteristic of lesions situated in the cerebral hemispheres. In cases where the involvement of the cerebellum predominates there may be produced the uncommon condition known as *acute cerebellar ataxia.* (See also, p. 383.) The initial symptoms are those of encephalitis in general, with the development sooner or later of such cerebellar manifestations as staggering gait, ataxia, and vertigo; often associated with nystagmus, disturbance of speech and tremor. I have reported[2] an instance of this very interesting condition, with statistics of 17 others from medical literature, and have since then seen 3 other cases.

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[1] Arch. of Ped., 1897, XIV, 809.

[2] Amer. Jour. Med. Sci., 1916, CLI, 24.

Encephalitis affecting the pons and medulla is observed oftenest in the course of acute poliomyelitis (Vol. I, p. 530), but may occur independently of this disease. After subsidence of the general early manifestations, there is discovered involvement of some of the cranial nerves with the symptoms depending upon this.

In the instances of multiple disseminated myelo-encephalitis, the result perhaps of some of the infectious disorders, the clinical characteristics are extremely variable, depending upon the regions principally affected. The final symptoms may be those of a multiple sclerosis, with disturbances of speech, diminished intelligence, convulsive conditions, and tremor.

Course and Prognosis.—The prognosis is always serious and uncertain. The great majority of cases occurring in the 1st year of life die after a short course. Subjects suffering from the more circumscribed lesions also may die in a very few days if the condition is severe. Very many milder cases recover, so far as the continuance of life is concerned, and some show no evidences of the disease later. The probability, however, is that symptoms of intracranial disorder of some sort will remain, oftenest those of infantile cerebral paralysis.

Diagnosis.—This is at first almost impossible. There are all the appearances of a meningitis, and this may, indeed, be the primary or an accompanying condition. The principle diagnostic characteristics are the acute febrile onset with general cerebral manifestations, followed soon by distinct localizing symptoms. In the form occurring in early infancy, the frequent failure of these localizing symptoms to develop leaves the diagnosis from meningitis always uncertain. The obtaining of a cloudy fluid by lumbar puncture points rather to meningitis. In the rapidly fatal cases of circumscribed encephalitis in older children diagnosis is also impossible, because the subjects die before localizing symptoms appear. In the disseminated myelo-encephalitis the distinction from multiple sclerosis rests principally upon the sudden febrile onset following upon some infectious disease, and the absence of a persistent increase of symptoms.

Cerebral embolism, sinus-thrombosis, and cerebral hemorrhage are to be differentiated from encephalitis by the absence of so rapid a development of high fever, and by associated conditions; yet the diagnosis in many cases is possible. (See pp. 340, 341, 343.) Abscess of the brain is of slower onset, and the fever is not so uniformly high. It is distinguished, too, by the later course of the disease (p. 348). Intracranial tumor lacks the sudden febrile onset. With all these conditions, however, mistakes readily arise and only careful study of all the symptoms and possible causes can lead to a probable diagnosis.

Treatment.—Since the onset is usually stormy and sudden, derivative measures may be first employed, such as free purgation, ice-bags to the head, hot mustard foot-baths, and the like. Means should also be used to control the fever. In older children leeches may be applied to the temples or behind the ears. Rest in bed and entire quiet are imperative. Care is to be taken that the child receive sufficient nourishment, even by gavage if necessary. Convulsive conditions or other evidences of excitement require the bromides and perhaps chloral. Warm tub-baths are often useful here. The treatment after the acute stage is over depends upon the symptoms which remain.

ABSCESS OF THE BRAIN

(Suppurative Encephalitis)

Etiology.—This disease is not common in early life, the majority of the cases occurring at a later period. Of 223 instances collected by Gowers[1] 24 were in the 1st decade and 48 in the 2d. Holt[2] could find but 27 reported cases in children under 5 years of age, to which he added the histories of 5 others. The disorder has more recently been discussed by Holmes.[3] Much the most frequent cause is extension from disease of the ear, usually chronic, or of the petrous bone or the mastoid cells. Another prominent etiological factor is trauma of the head, with or without fracture. Among causes less frequently observed are diseases of the bones of other parts of the head, or infectious emboli from a suppurative process elsewhere in the body. These last mentioned are, however, very unusual in early life. In a few instances the fungus of thrush has been reported present, as by Ribbert[4] and others; and in a number no cause whatever can be discovered.

Pathological Anatomy.—When the result of distant causes the lesions are usually multiple. In the large majority of cases, however (80 per cent., Gowers) abscess depends upon local conditions and is single. It may then occupy various parts of the brain, oftenest the tempero-sphenoidal lobe, but also frequently the cerebellum or the frontal lobe. Other regions are seldom the seat of the lesion. The locality depends largely upon the nature of the primary disease. Traumatic abscess is generally in the cerebrum; otitic abscess here or in the cerebellum; and that depending upon the disease of the orbit or of the nose usually in the frontal lobe. Körner[5] estimates that abscess in the cerebrum is more than twice as frequent as in the cerebellum, and in Holt's series of 32 cases in children the cerebellum was involved in but 8 instances.

The process begins as an area of red softening in which pus cells multiply, the abscess rapidly increasing in size and finally containing thick or thin greenish-yellow pus of acid reaction and sometimes of a fetid odor. Various species of bacteria may be present. The final size ranges from that of a pea up to an accumulation filling the greater portion of a hemisphere. The shape is round or irregular. Many abscesses remain always without a capsule; others begin to form one in the 2d or 3d week, and develop this completely by the end of 2 months, by which time it is composed of firm, thick, fibrous tissue with a smooth inner lining. The surrounding brain-tissue is edematous and softened. An abscess may cause death through its pressure interfering with the functions of the brain; may rapidly increase in size and produce a purulent meningitis; or may rupture in various directions, oftenest into the ventricle, less often upon the surface of the brain, or occasionally through the ear or nose.

Symptoms.—These are very varied; often obscure; and necessarily often combined with and confused by the presence of the symptoms of the primary disorder. They are both general and focal in nature; the latter often absent. Ordinarily they are classified as of three stages, which, however, are not always clearly characterized or sharply distinct from each other or present in every case.

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[1] Diseases of the Nervous System, Amer. Edit., 1888, 852.
[2] Arch. of Ped., 1898, XV, 81.
[3] Arch. of Intern. Med., 1916, XVII, 591.
[4] Berl. klin. Woch., 1879, XVI, 617.
[5] Die otitische Erkrank. des Hirns, 1903, 3. Aufl., 139.

1. The Initial Stage.—This often commences insidiously and is unsuspected; but the intensity and rapidity of development of the initial symptoms may vary from this up to the severe cases with very abrupt and stormy onset. The initial stage when well marked is characterized by headache which may be localized; vomiting; prostration; chills; and fever of a continuous type and of moderate severity, not often with delirium. There are sometimes general or local convulsions. This is the period of inflammation and marks the formation of the abscess. In cases of acute abscess the advance from this stage is directly to the terminal one with its fatal ending. In chronic abscess, however, the initial period lasts from a few days to 3 or 4 weeks and is then followed by the stage of latency.

2. The Latent Stage.—During this all symptoms disappear, or there remain at most occasional headache and rises of temperature; chills at irregular intervals; a degree of somnolence or of mental disturbance; and in some cases convulsions. Focal symptoms which have appeared in the first stage will persist. The duration of latency is extremely variable. It may be of some weeks or sometimes a very much shorter time, and occasionally even last for several years.

3. The Terminal Stage.—This begins gradually or suddenly, and marks the rupture of the abscess or the extension of the inflammation to surrounding regions. When the initial stage has been very mild, the symptoms of the third stage may be the first to attract notice. When, too, the period of latency has been absent, as in the acute cases, there is no sharp demarcation between the initial and terminal stages. The third stage is characterized by a return of the early symptoms in increased force. The fever is high and continuous; the headache often very severe and persistent; symptoms of meningitis develop; the pulse is irregular and either slow or rapid; the respiration may be Cheyne-Stokes in type; vomiting and vertigo may occur; there are often delirium, convulsions, paralysis, and optic neuritis; and finally the patient passes into coma. The duration of this stage is usually not more than a few days.

In addition to the symptoms described, there are not infrequently distinct *localizing symptoms*, which may indicate the situation of the inflammation. This is, however, much less often observed than in the case of tumor. It is of common occurrence that even large abscesses may be present without any such symptoms appearing. This is due to the fact that the regions oftenest affected by abscess are not those which can produce focal symptoms. Headache when localized is most frequently upon the side of the head in which the abscess is situated. If occipital it is an indication that the lesion is in the cerebellum. If the abscess is in the motor area paralysis of various sorts or localized convulsions may occur, corresponding to the centres involved. Vomiting and giddiness are often seen in cerebellar abscess but are frequent in the cerebral cases also. Slow and difficult speech, ptosis, and disturbances of the occular movements point to involvement of or pressure upon the pons. Local tenderness of the scalp may indicate the position of a superficial abscess.

Course and Prognosis.—The description given of the duration of the various stages is far from constituting a universal rule. In many of the acute cases the course is much more rapid than this, and in half of the cases collected by Gowers[1] the total duration did not exceed 2 weeks.

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[1] *Loc. cit.*, 865.

The terminal stage sometimes appears suddenly, and its symptoms may be the first to be noticed, owing to the slight development of those of the initial stage. In some cases death may take place unexpectedly from convulsions, or the end come rapidly when rupture of the abscess has occurred with the immediate development of severe meningitis.

The prognosis is always very grave, nearly all the patients dying unless operation gives relief. Exceptionally in the cases with very long latency the wall of the capsule may become calcified and the contents dry and harmless.

Diagnosis.—The principle diagnostic symptoms are the slow onset with gradual increase of headache, the presence of fever, and the development later of convulsions and coma. These, combined with the existence of focal symptoms, and especially with that of such possible causes as otitis or trauma, will be sufficient in many instances to make a diagnosis possible. The occurrence of the symptoms in stages is of great diagnostic assistance when these can be recognized. Yet in spite of such aids, the diagnosis is often most difficult and frequently impossible. Trauma or otitis may be productive of meningitis as readily as of abscess, and a chronic abscess may sometimes simulate tumor in many respects.

Meningitis causes perhaps the greatest diagnostic difficulty. It is more rapid in onset and shorter in course; the pulse is oftener accelerated than in abscess; the initial fever is higher; there is greater tendency to rigidity and convulsions, although the latter are less often localized; leucocytosis is more decided; pain in the extremities and back is frequent, and focal symptoms are not common. To all this, however, there are numerous exceptions. Lumbar puncture reveals a clear fluid in abscess, and pus in the purulent forms of meningitis.

Serous meningitis depending upon otitis or trauma is often distinguished with the greatest difficulty, since the primary cause might produce either affection, and optic neuritis may occur in either. The principal diagnostic characteristics of this form of meningitis are its more sudden onset and often its rapid disappearance. The examination of the cerebrospinal fluid obtained by lumbar puncture does not certainly aid; but the operation often gives relief to the symptoms in meningitis but not in abscess. *Intracranial tumor* has no initial febrile stage; choked discs are more uniformly present; focal symptoms more constantly seen; and there is no rapidly developing febrile terminal stage. *Sinus-thrombosis* shows a more rapid production of high and irregular fever; rigors are more common, with the evidences in general of sepsis; and focal symptoms are absent. The diagnosis of many of the conditions which simulate abscess is rendered still more difficult by the fact that this disease may be combined with them as a sequel or a cause.

Treatment.—The only palliative treatment is that suitable for any cerebral inflammation; such as rest in bed, ice to the head, purgatives, and remedies to quiet symptoms of nervous excitation. The only really applicable curative treatment is surgical, and this is often completely successful, and should be employed in every case when the symptoms indicate the position of the lesion. Oppenheim[1] reports 104 collected operative cases of otitic cerebral abscess with 48 recoveries; and 30 situated in the cerebellum and operated upon with 12 recoveries.

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[1] Nothnagel, Handbuch. d. spec. Path. u. Therap., 1896, IX, 1, 3, 3, 253.

TUMORS OF THE BRAIN

Etiology.—As with intracranial neoplasms at any age, the causes in childhood are entirely unknown, with the exception of those of a tuberculous or syphilitic nature. The growth may be primary in the brain, or secondary to similar formations elsewhere. As regards the influence of age, rather the larger number occur in childhood and early adult life; but they may be seen even in infancy, although they are rare in the 1st year and not frequent in the 2d. In 650 cases collected by Gowers[1] 18.5 per cent. occurred in the 1st decade, and 14 per cent. in the 2d.

Nature and Position.—The growths found in early life are not of numerous sorts. According to M. A. Starr,[2] the cerebellum and the cerebral axis (basal ganglia, internal capsule, corpora quadrigemina, crura cerebri, pons, and medulla) are the portions oftenest affected in childhood, while the cerebral cortex is that most frequently involved in adults.

This author has analyzed 300 collected cases seen in childhood and youth, and 300 others occurring in adult life. The relative incidence of the different varieties of morbid growths, and the situation oftenest occupied is shown in the following table given by him.[3]

Table 87.—Brain-tumors in Children and Adults

Situation	Tuberculous		Gliomatous		Sarcomatous		Gliosarcomatous		Cystic		Carcinomatous		Gummatous		Not stated		Total	
I. Cerebral axis;																				
1. Basal ganglia and lateral ventricles..	14	3	3	9	5	8	..	..	1	1	1	2	..	1	3	5	27	29
2. Corpora quadrigemina and crura cerebri...........	16	1	1	2	3	2	..	5	..	.	..	1	..	..	1	7	21	18
3. Pons............	19	11	10		5	1	2	1	1	.	..	2	..	3	1	..	38	18
4. Medulla..........	2	..	..		..	..	..	..	1	.	2	..	..	..	1	..	6	1
5. Base.............	...	3	..	2	1	3	1	1	1	.	1	..	..	..	4	1	8	10
6. Fourth ventricle...	1	..	1	..	1	1	..	..	..	.	1	2	..	..	1	1	5	4
II. Cerebellum..........	47	8	15	8	10	13	1	6	9	.	3	..	..	..	11	10	96	45
III. Multiple tumors.....	34	4	..	2	3	5	..	..	2	.	..	2	1	3	3	1	43	17
IV. Cortex cerebri.......	13	9	6	19	1	46	..	8	..	1	1	19	..	13	..	12	21	127
V. Centrum ovale.......	6	2	1	11	5	7	1	4	15	.	1	3	1	..	5	4	35	31
	152	41	37	54	34	86	5	25	30	2	10	31	2	20	30	41	300	300

The first columns are children's tumors; the second columns adults' tumors.

Tuberculous tumors are the variety much most frequently seen in early life. Growths of this sort are probably always secondary; oftenest multiple; and especially liable to be found in the cerebellum, and next oftenest in the pons. The tumor is from the size of a pea up to that of a walnut, or larger. It is observed sometimes in infancy, but most frequently in later childhood. According to Gowers[4] 25 per cent. of the cases occur in the first 20 years of life, and half of these in the first 10 years. The central portion of the tumor is often cheesy, and a localized meningitis is nearly always associated. *Glioma* is somewhat less common than in adult life, although seen even in infancy. It is always primary; of various size, being sometimes miliary, sometimes very large; and much oftenest single. It is composed of neurogliar tissue and is very vascular, and not sharply defined. The cerebral hemispheres, the cerebellum, and the pons are favorite seats, but it may occur in any part of the brain. *Sarcoma* is either primary or secondary; generally single; and of varying size and rapid growth. Any part of the brain may be involved, and any age affected; but the pons, the cerebellum, and the cortex, are favorite situations. Carcinoma, gliosarcoma, fibroma, gumma, and growths of other nature are all of rare occurrence in early life. Cysts of a parasitic nature are very rarely encountered in the United States. The tendency to cystic degeneration may be observed in some varieties of tumors, as in sarcoma and glioma. Secondary changes may be seen in neighboring parts of the brain; such as localized meningitis and the conditions resulting from pressure. Among the latter are necrosis of the adjacent brain-substance; interference with the circulation; flattening of the convolutions; hydrocephalus; and erosion of the bones of the skull.

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[1] Diseases of the Nervous System, Amer. Edit., 1888, 869.

[2] Brain Surgery, 1893, 202.

[3] There was an error in the addition of the figures in the original, which I have corrected with Dr. Starr's permission.

[4] *Loc. cit.*, 870.

Symptoms.—These may be divided into *general* and *local*.

General Symptoms.—Headache is one of the earliest and most important manifestations. It may be paroxysmal or continuous; diffuse or localized; and intensely severe or slight, or exceptionally even absent. With this there is sometimes localized tenderness of the skull. Vomiting is a common symptom seen in much over half of the cases. It appears to be independent of any evidence of indigestion, loss of appetite, or the taking of food, and nausea may be absent. The vomiting is forcible, most frequent when headache is present, and is among the earlier symptoms, often disappearing later in the disease. General convulsions with unconsciousness may develop at any period of the course; be severe or only slight; and be seen only at long intervals or be of frequent occurrence and often grow more so as the disease progresses. An aura may be witnessed as in idiopathic epilepsy. A spasm limited to one region may precede the general convulsions, and is then of importance among localizing symptoms. Optic neuritis, nearly always bilateral, and followed finally by atrophy, is one of the most important general symptoms, absent in only about 10 per cent. of the cases. This is an early development especially in tumor of the cerebellum, base of the brain, or crus. Vertigo is an early symptom not infrequently present, either constant or produced only when the child moves. Slowness of the pulse is inconstant, and is often temporary or absent. Irregularity or slowness of respiration is a symptom occasionally witnessed, oftenest in an advanced stage of the disease. Psychic disturbances of a varied nature are frequently present. There may be irritability and fretfulness, or the disposition may be altered in some other way; or apathy, somnolence, insomnia, dreaming, occasionally a delirious state, or mental deficiency may be observed.

Local Symptoms.—These are by no means always present. They are necessarily varied, in keeping with the situation of the tumor, the rapidity of its growth, and the existence of other conditions, such as a complicating meningitis or the results of irritation or of pressure. Yet tumors in the same situation do not always produce identical symptoms. The subject is more appropriately discussed in treatises upon cerebral localization, and only a brief outline can be given here.

Growths of the *motor area* produce hemiplegic or monoplegic symptoms in the arm or leg or on one side of the face, dependent upon the convolutions involved. Later the paralysis may extend as the tumor grows. Localized anesthesia may be found. Convulsive movements in the region showing paralysis are common and usually antedate this. They are often of the Jacksonian type without loss of consciousness, but sometimes the convulsions are general and epileptiform. Conjugate deviation of the eyes is common. The meninges are frequently secondarily involved with the production of the usual symptoms. If the growth is in the *frontal lobe,* of rare occurrence in children, there may be few symptoms or none at all. Convulsions may occur from an extension of the irritation, but generally without paralysis. Motor aphasia is produced by a growth in the third frontal convolution on the left side. A frontal ataxia has been reported, with disturbance of gait and station. Exophthalmus on the side of the lesion has been described. Tumors of the *temporo-sphenoidal lobe* are uncommon. They may give rise to sensory aphasia and disturbances of hearing, and sometimes of the senses of smell and taste. Those of the convolutions of the *Island of Reil* cause aphasia, if the lesion is on the left side. Growths of the *parietal lobe* are extremely rare in childhood. They produce no distinct localizing symptoms. Any manifestations present, except those of a general character, are of a sensory nature, such as disturbances of the sense of touch and of the muscle-sense, ataxia, and word-blindness. Hemiparesis and Jacksonian convulsions may be produced secondarily by pressure upon the motor region.

Involvement of the *occipital lobe* is very infrequent. It produces homonimous hemianopsia, or sometimes other disturbances of vision. Tumors of the *centrum ovale,* not common in children, are liable to cause symptoms similar to those of the corresponding cortical region, except that convulsions are infrequent. If the growth is more deeply situated it may press upon the basal ganglia and give rise to the symptoms incident to this. Growths involving the *basal ganglia,* of comparatively common occurrence in childhood, have no distinctly localizing symptoms Convulsions are not frequent; in sharp contrast to lesions of the cortex. The nature of the symptoms depends upon which of the conducting fibres from the cortex are compressed. If the internal capsule is pressed upon, hemiplegia follows. With tumors of the region of the optic thalamus there is hemianesthesia, hemiathetosis, or hemichorea. There is frequently involvement, too, of the functions of the cranial nerves, and speech may be affected. Tumors of the *corpora quadrigemina* cause paralysis of the ocular muscles, affections of hearing, tremor, ataxia, and nystagmus. Lesions of the *crura cerebri* occasion paralysis of the oculomotor nerve on the side of the lesion; shown by ptosis, external strabismus, loss of the pupillary reflex, and dilatation of the pupil. With this is combined hemiplegic paralysis, including the face, upon the side opposite the lesion. There are also sometimes tremor and ataxia. Hemianesthesia may sometimes be present; convulsions are uncommon.

Tumors of the *pons* are of comparatively frequent occurrence in early life and seen oftener than in adults. They produce a crossed paralysis, involving the cranial nerves on the side of the tumor and the extremities on the other side, sometimes with loss of sensation. Which cranial nerves are affected depends upon the position of the growth. If the lesion is high in the pons the cranial nerves escape. If somewhat lower there is involvement of the 3d and 5th nerves, with paralysis of the ocular muscles as a very prominent symptom of the former and with facial neuralgia and sometimes ulceration of the cornea as indications of the latter. Derangement of these two nerves is usually present in cases of pontine tumors. If the lower portion of the pons is involved, the 6th, 7th and 8th nerves are affected. There is then especially facial paralysis; disturbances of taste; and sometimes deafness. The general symptoms are frequently much less marked than are the local in growths in the pons. Convulsions are not common, but there is often rigidity of the extremities with increased reflexes; and this may be diplegic in type, if the tumor occupies both sides of the pons. Tumors of the *medulla*, not common in children, affect the last four of the cranial nerves, and often by extension some of the other cranial nerve-nuclei as well. The principle symptoms are dysphagia; deafness; difficulty in articulation; and involvement of the tongue. There is also often more or less severe disturbance of the action of the heart and of respiration; flushing; and sweating. Polyuria and glycosuria have been reported. There is frequently also at first a unilateral and later a bilateral involvement of the pyramidal tracts, with rigidity and increase reflexes; or the sensory tracts may be affected with consequent ataxia and anesthesia.

Tumors of the *cerebellum*, oftener seen here in childhood than in any other situation, give rise to symptoms when the middle lobe is diseased, or when a tumor in one hemisphere is large enough to affect this lobe by pressure. The most characteristic indications are cerebellar ataxia, vertigo, and nystagmus; combined with an early development of marked general symptoms, such as headache; projectile vomiting; optic neuritis, and rigidity of the neck. Blindness may develop. The ataxia may be so great that the gait is that of deep intoxication; or the patient be entirely unable to keep from falling. Other symptoms may arise, depending upon the pressure of the cerebellar growth upon neighboring parts; the various cranial nerves suffering, or the pyramidal tracts being compressed with consequent weakness in, and increased reflex excitability of, the extremities. There is occasionally opisthotonus but seldom convulsive movement.

Tumors of the *pituitary* and *pineal glands* give rise respectively to symptoms of a peculiar nature which will be discussed elsewhere in connection with the Ductless Glands and Internal Secretions (pp. 522, 523, 533).

Course and Prognosis.—In general the course is slowly progressive, although not at a uniform rate. Periods of intermission may sometimes occur during which the condition remains unchanged, these being followed by exacerbations of the symptoms; while in other cases there may be a rapid and continuous increase in the severity of these. This is especially true if a complicating meningitis develop. The duration of the disease depends upon the nature of the growth. Soft sarcomata and gliomata grow rapidly, and the course is apt to be shorter. The tuberculous tumor is of longer duration unless the development of a complicating tuberculous meningitis cuts the disease short. The situation, likewise, is of importance. A tumor in the frontal lobe, for instance, is liable to produce trouble to a much less extent than one involving the pons or the medulla.

The duration varies from several months to several years, but is usually within 6 months to 2 years. Very exceptionally the whole course may be but a few weeks. The disease is nearly always fatal, death ensuing as the direct result of it, or through some complication, such as meningitis or hydrocephalus. Sometimes the end comes suddenly; oftenest it takes place in coma with exhaustion. It would appear possible for cases of tumor of the brain to recover without operation. Probably this is true of many cases of gumma where the patient has received appropriate treatment. Tuberculous tumors may become encapsulated, and partial recovery, or at least an arrest of the disease, take place. This event is, however, very uncommon.

Diagnosis.—This rests especially upon the slow onset; progressive course; the existence of such general symptoms as headache, vertigo, optic neuritis, vomiting, convulsions and mental disturbance; and the presence of various localizing symptoms as described. These latter are of more diagnostic value than the general manifestations, with the exception of optic neuritis, which is very suggestive but which may occur in many other conditions. Lumbar puncture may be employed as a diagnostic procedure, but is not without danger. Only a small amount of fluid should be withdrawn, with care not to diminish intracranial pressure too greatly. Radiology is not often of any considerable aid.

The nature of the neoplasm can only occasionally be surmised. Glioma is the variety most rapid in onset; most liable to exhibit exacerbations and remissions; and most likely to produce sudden apoplectiform seizures during the course of the case. A tuberculous growth is that oftenest with the slowest onset and most prone to exhibit a long stationary period. Tumors in the pons or cerebellum are likely to be tuberculous or gliomatous.

The various symptoms indicating the situation of the tumor have already been pointed out. Among the diseases most readily confounded with tumor are abscess of the brain, chronic hydrocephalus, tuberculous meningitis, chronic basilar meningitis, and the cerebral form of poliomyelitis. *Cerebral abscess* is differentiated by the existence of a definite primary cause; a more rapid and irregular course, often with its characteristic stages; a more infrequent occurrence or a later development of choked discs; less intense headache; the presence of fever and chills; less common involvement of the cranial nerves; and a much lesser frequency of localizing symptoms. *Chronic hydrocephalus* does not often cause diagnostic confusion, excepting as regards the possibility of its having developed as a secondary condition. The history of a primary hydrocephalus is entirely different from that of tumor. *Tuberculous meningitis* can only be confounded with tumor when it runs an exceptionally slow course. Optic neuritis is, moreover, much less common and less marked, and rigidity of the neck more frequent. Localizing symptoms less often appear, and some degree of fever occurs. Lumbar puncture gives a fluid which is often slightly cloudy, exhibits evidences of inflammation, and may show an increase of lymphocytes together with tubercle bacilli; while in tumor the fluid is of normal character. In both conditions it may be under increased pressure. Yet in spite of these facts the diagnosis may exceptionally be extremely difficult, and I have seen the failure to distinguish the two conditions made by expert neurologists. *Chronic posterior meningitis* may strongly suggest tumor of the base of the brain. It occurs, however, chiefly in infancy, and exhibits usually marked opisthotonos, rigidity of the neck, and bulging of the fontanelle. *Poliomyelitis of the cerebral type*, or *encephalitis* from any cause, could suggest brain-tumor with hemiplegic paralysis. There is, however, a sudden febrile onset and the course of the case is entirely different.

Treatment.—The only medicinal treatment is antisyphilitic in cases suspected of being due to this disease. In all other cases an early surgical intervention should be obtained if the situation of the tumor permits of this. The operation, it is true, is a most serious one; but the alternative is still more so. Tumors of the motor region offer a very good chance of successful removal. Those situated elsewhere are less favorable; and in some regions, as in the cerebral axis, or where the tumors are multiple, operation cannot be done. Unfortunately the position of most tumors renders operation rarely possible. Even in these, however, operation for decompression may relieve the symptoms for a time, delay the loss of sight, and prolong life.

Palliative treatment consists in the relief of nervous symptoms present, especially headache.

HYDROCEPHALUS

Although of various forms, depending upon different etiological factors and to some extent upon diverse pathological conditions, all varieties may be conveniently studied together. It is customary to divide the disease into *External Hydrocephalus* and *Internal Hydrocephalus*, the latter being again subdivided into the *Acute* and the *Chronic* forms. The condition in all consists in an accumulation of serous fluid within the cranium.

1. EXTERNAL HYDROCEPHALUS

In this chronic form of hydrocephalus there is an excess of fluid in the subdural space. It is an uncommon disorder, occurring as a sequel to pachymeningitis, seen in the course of chronic meningoencephalitis and serous meningitis, or encountered where there has been either wasting or lack of development of a portion of the brain (*hydrocephalus e vacuo*). The fluid is clear or sometimes blood-tinged, and the amount generally small; but sometimes it is large and the skull grows progressively more distended. There is no evidence of inflammation of the membranes found at autopsy. Sometimes an external hydrocephalus is combined with one of the internal variety, or may be produced by the rupture of one of the latter form into the subdural space.

The **symptoms** are at first those of the primary causative disorder. Later the amount of fluid may be not large enough to produce any characteristic manifestations; but in the cases with progressive increase of the quantity the symptoms are those seen in chronic internal hydrocephalus.

2. ACUTE INTERNAL HYDROCEPHALUS

The title is applied synonomously both to tuberculous meningitis and to the cases of serous meningitis in which the ventricles are especially involved. The course is not a prolonged one and the amount of fluid in the ventricles is not large. The symptoms have already been sufficiently described.

3. CHRONIC INTERNAL HYDROCEPHALUS

This variety is the one much most frequently seen. It may be divided into *congenital* and *acquired*. By the term congenital is not meant, however, that the condition is necessarily noticeable at birth, although this may be the case; but that the anatomical conditions producing it are then present.

Etiology.—The etiology of *congenital* cases is not understood. This form of the disease is probably due to some abnormality of development, and is not infrequently associated with other malformations, such as spina bifida, harelip, and club-foot (Fig. 348). It may be perhaps the result of an earlier intra-uterine meningitis. A familial tendency is sometimes observed. Among the causes of chronic *acquired* hydrocephalus may be mentioned cerebrospinal fever, abscess or tumor of the brain, chronic posterior meningitis, trauma, and syphilis. The exact influence of syphilis is as yet undetermined. It would appear to have no causative connection in decidedly the majority of cases. Rachitis has been believed to be a factor in some instances. Certainly in many rachitic heads there appears to be a tendency to an unusual accumulation of fluid. This, however, is but temporary. In the domain of trauma may be included the effect of operation upon spina bifida, which not infrequently is followed by hydrocephalus, or the increase in the symptoms in cases where this is already present.

The method of production of hydrocephalus has received much attention, and many different views have been held. The investigations of Dandy and Blackfan[1] have modified former opinions to some extent. According to their studies the cerebrospinal fluid is secreted by the choroid plexuses in the different ventricles. It finds its exit through the aqueduct of Sylvius into the fourth ventricle, and thence through the foramina of Magendie and of Luschka into the subarachnoid space. It is absorbed from the entire subarachnoid space. The Pacchionian bodies possess no power of absorption, contrary to the previously accepted view. Under normal conditions there is a balance between production and absorption. This balance may be disturbed, and hydrocephalus be produced. Practically the same results have been obtained by Frazier and Peet.[2] It becomes evident, then, that hydrocephalus may be produced in one of several ways: Either there may be a hypersecretion from the choroid plexuses; or a diminished absorption from the subarachnoid space, dependent upon inflammatory lesions of the meninges; or mechanical obstruction to the exit of fluid from the ventricles.

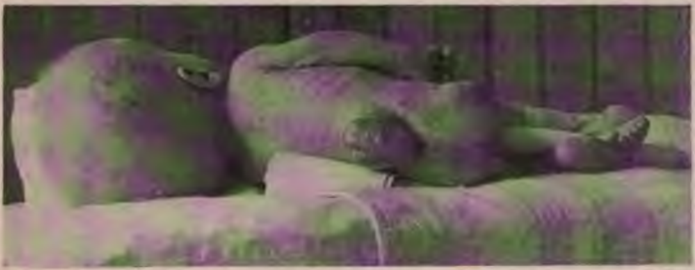

FIG. 348.—HYDROCEPHALUS, SPINA BIFIDA AND CLUB-FOOT.
From a patient in the Children's Hospital of Philadelphia.

Pathological Anatomy.—The characteristic lesion is a dilatation of the lateral ventricles with a corresponding thinning of the cortex of the hemispheres. The third and fourth ventricles may also be distended, as well as the passages connecting the ventricles, this depending upon the nature of the cause. The amount of fluid varies from a few ounces only up to ½ pint (237.) and exceptionally a quart (946.) or more. In the primary non-inflammatory cases the fluid is usually clear, contains no cells and but a very little albumin, and is of low specific gravity. When inflammation is the cause of the disease it may be turbid and is richer in albumin.

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[1] Amer. Jour. Dis. Child., 1914, VIII, 406.
[2] Amer. Journ. Physiol., 1914, XXXV, 268.

A purulent character occasionally results through infection. When the amount of fluid is not large, as is oftener true of acquired hydrocephalus, only certain of the ventricles may be distended, varying with the case. When in very large amount, as characteristic chiefly of congenital hydrocephalus, the convolutions are much flattened and thinned; and in such cases the brain substance of the hemispheres may even appear macroscopically to be entirely destroyed, the fluid being contained in two membranous sacs (Fig. 349). Through the pressure the basal ganglia become flattened and the cerebellum likewise, and the velum interpositum and septum lucidum disappear. The choroid plexus may be of natural appearance, or thickened, enlarged, firm, and unduly vascular. The walls of the ventricles may be normal or irregularly thickened, and they and the meninges may show evidence of inflammation in acquired hydrocephalus. The aqueduct of Sylvius and the foramina of Luschka and of Majendie may be closed, and the capacity of the cisterna magna much diminished. The bones of the cranium are greatly altered in cases beginning early. They become much thinned and translucent; the sutures are very wide; the fontanelles much enlarged; the roof of the orbit depressed. Naturally all the changes described vary in degree, depending upon the amount of fluid and the

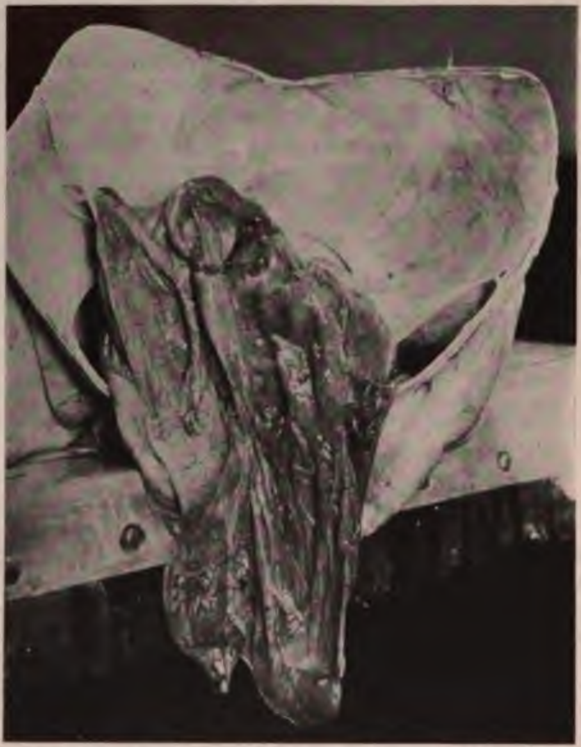

FIG. 349.—OPENED SKULL IN HYDROCEPHALUS.

Same case as in Fig. 350. On removing the calvarium a gush of liquid occurred and a collapsed sac of thin membrane remained, all cerebral tissue above the basal ganglia seeming to have disappeared.

pressure exercised by it. If hydrocephalus is acquired after the bones of the head have become ossified, either prematurely or normally, the skull exhibits no alteration in character, or at the most a slowly developing and moderate increase in size.

Symptoms.—In the most common variety, the *congenital form*, the head may be so much enlarged before birth that labor is interfered with, and in the worst cases perforation of the skull may be required. More often, however, the head is but little, if any, larger than normal at birth, and no change is noticed for a period varying from days or weeks often up to 2 or 3 months. Then it will be observed that the growth of the cranium in all directions appears to be abnormally rapid. In well-developed cases the head has a shape approaching the globular; the parietal regions may project beyond the ears, and the forehead is overhanging with very prominent frontal eminences (Fig. 350). The fontanelles are wide-open and bulging; the sutures wide, and often with Wormian bones situated in them. The veins of the scalp are prominent and the hair scanty. The cranium may be translucent and fluctuate readily in severe cases, and there may be a somewhat tympanitic note on percussion. The face by comparison appears unusually small. The depression of the roof of the orbit causes a corresponding downward displacement of the eyeballs, the sclera showing above the iris, and the lower part of this latter or even a portion of the pupil being covered by the lower lid. There is sometimes nystagmus, strabismus, sluggishness of the pupils, or optic atrophy. The external ear is somewhat depressed. The degree of distention of the head is very variable. A circumference of from 50 to 70 cm. (19.7 to 27.6 inches) is not uncommon, and even 100 cm. (39.4 inches) has been reported.

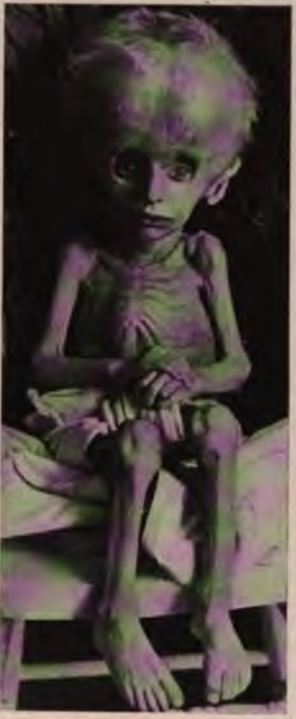

FIG. 350.—HYDROCEPHALUS.

From a patient admitted to the Children's Hospital of Philadelphia, Nov. 17, aged 13 months. Head large at birth, and continued to increase in size. Examination showed the circumference 22½ inches (57 cm.), veins dilated, sclera visible above iris, arms and legs usually rigid and with tremor, no optic neuritis. Died Jan. 3.

In addition to these alterations of the head there appear sooner or later failure of satisfactory development of the body with general weakness; inability to hold the head erect; slowness in learning to sit up or to walk; and either paresis of a flaccid type, or a tendency to spasticity with marked increase of the reflexes and clenching of the hands. Convulsions are not uncommon. The digestion may be disturbed, and much emaciation develops as the case advances. The intelligence may long be normal, but oftener there is some degree of apathy, dullness, irritability, imperfect speech, and finally often idiocy. It is wonderful, however, in cases in which the advance has been slow, how little the intellect may be affected for a long period. One sees children with a degree of intelligence quite remarkable, considering the very small amount of brain-matter in the hemispheres, as revealed at autopsy.

In the *acquired cases* the symptoms are usually less marked, if ossification has not yet occurred, and the head may increase in size but little, owing to the small amount of fluid which accumulates in the ventricles. After ossification has taken place, a small quantity of fluid may produce by its pressure as severe general symptoms as is observed in advanced congenital cases with a large amount present. The symptoms in this form are variable and uncharacteristic; among them being optic neuritis; headache; vomiting; coma; and other evidences of compression. Contractures and rigidity are often among the most noteworthy symptoms.

Course and Prognosis.—The course is extremely variable. Some cases advance rapidly to a fatal ending within a few weeks after birth; others slowly but steadily for a time and finally stop spontaneously, the head ceasing to grow in size, and no increase in general symptoms developing, or even improvement taking place. Such cases, not numerous, may reach adult life. Others cases exhibit periods of temporary cessation of advance alternating with exacerbations.

The prognosis is on the whole unfavorable. Only the mildest cases offer any great hope of recovery. Those depending upon syphilis are to a degree susceptible of benefit by antisyphilitic treatment, Hochsinger[1] having had 16 recoveries in 35 cases. The acquired hydrocephalus dependent upon cerebrospinal fever presents usually an unfavorable prognosis. The large majority of cases of congenital hydrocephalus die in the early years of life, and most of them in the 1st year. The cause of death is either progressive wasting and exhaustion, or some intercurrent complication. In general the duration of life is dependent upon the rapidity of the accumulation and the degree of yielding of the bones of the skull.

Diagnosis.—In cases with distinct enlargement of the head the diagnosis is easy, since the appearances are so characteristic. Rachitis is the disease causing most confusion in these cases, especially as it is not uncommon for temporary hydrocephalic accumulation to be associated with rickets. Rickets, however, is distinguished by the peculiarly square shape of the head; the deposition of bone on the frontal and parietal eminences and about the fontanelles; the failure of the head to increase rapidly in size; and the evidences of the disease elsewhere in the body. The suspicion of hydrocephalus is sometimes aroused, too, in the case of children in the 1st year who have abnormally large heads with prominent foreheads, but in whom this is merely an individual peculiarity. The absence of widely distended sutures and fontanelles and of abnormally rapid growth of the head will soon remove all doubt. (See Normal Growth of the Head, Vol. I, p. 32.) In acquired cases in older children, with well-advanced ossification of the bones of the cranium, the diagnosis of hydrocephalus is often impossible, except from the knowledge of the nature of the primary cause. The condition closely resembles tumor.

The diagnosis of the nature of the cause is a matter of interest. As shown by Dandy and Blackfan,[2] if a solution of phenolsulphonephthalein is injected into the ventricles in cases of obstruction it enters the spinal subarachnoid not before from 30 to 50 minutes (normal time 5 minutes almost invariable in normal cases), and several days are required before it is completely eliminated by the kidneys. On the other hand, when the difficulty is one of absorption, the fluid almost immediately passes into the spinal subarachnoid, as shown by lumbar puncture, but is absorbed very slowly from it, and the elimination by the kidneys is much retarded. Injection into the subarachnoid space of the cord is also followed by delayed absorption and elimination in cases of the latter class.

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[1] Studien über hereditäre Syphilis, 1904, Theil 2, 506; 516.

[2] *Loc. cit.*

Treatment.—None of the various methods of treatment by drugs proposed in the past have proven of any value, except the employment of mercury and of the iodides in cases dependent upon syphilis. Certainly in all instances presumably syphilitic this treatment should be tried, and even in those probably not of this nature the effort to aid in this way should be made. The administration of thyroid extract has been urged by Frazier[1] in cases dependent upon hypersecretion, on the ground that this substance has a distinctly inhibitory effect upon the secretion from the choroid plexus. External pressure of the head, as by bandages, has been advocated in the hope of inducing absorption. It must be done guardedly if at all. I have never seen any benefit follow. Various operative measures have been proposed. Among them lumbar puncture is employed to relieve the intracranial pressure. This it will sometimes do if there is no mechanical obstruction existing in the passages from the ventricles to the spinal subarachnoidal space; and by its frequent repetition undoubted relief is sometimes given; but this is usually only temporary. A trial of it should, however, certainly be made. The same is true of puncture of the ventricles when lumbar puncture does not avail. Various efforts have been put forth to establish permanent drainage of the ventricular fluid into the subarachnoid space, the subcutaneous tissue, or the jugular vein. There have been a few remarkable cases of improvement, but the operations are serious ones, and the mortality is high. Puncture of the corpus callosum is probably one of the simplest and best operative procedures. All other treatment is supportive and symptomatic.

ATROPHY AND SCLEROSIS OF THE BRAIN

1. General or Local Atrophy of the Hemispheres.—This condition as encountered in early life is always a secondary process or a malformation. It may be seen, for instance, in the porencephalus and the agenesis corticalis occurring as congenital affections (pp. 309, 310), or may result later from compression by a hydrocephalic effusion. As a more local process it may be due to the pressure exerted by tumors or to a meningo-encephalitis, perhaps of a syphilitic nature, or may be the result of hemorrhage. It is seen, too, as a sequel to conditions of long-continued or extreme exhaustion, such as marantic states produced in any way. Atrophy of the nervous element is commonly combined with a certain degree of sclerosing process, exhibited in the increase of the connective tissue. The affection may involve only a few convolutions, or a larger portion of one or both hemispheres. The basal ganglia may share in the process, or the cerebellum to some extent; and secondary degeneration may follow in the pyramidal tracts of the spinal cord. The primary symptoms are those of the original disease; and those developing later are to be classed among the manifestations of infantile cerebral paralysis (p. 365).

2. Tuberous Sclerosis.—The uncommon affection described under this title by Bourneville,[2] the cause of which is unknown, is characterized

[1] Amer. Jour. Dis. Child., 1916, XI, 95.

[2] Arch. de Neurol., 1880, I, 81.

anatomically by the development of rounded, pea-sized or larger, nodular masses scattered upon the cortex, extending into the gray-matter beneath, and occurring also within the ventricles. They consist chiefly of hypertrophy of the glial tissue, with cells believed to be ganglion cells. The disease is probably a developmental disturbance, the lesions beginning to appear soon after birth or even before it. The **symptoms,** too, are observed soon after birth in most instances, the most marked one consisting in a great diminution of the intellegence even to the degree of idiocy. Convulsions generally appear in the early months of life and are frequently repeated. Paralysis, with or without contractures, may develop. The diagnosis rests upon the combination of these symptoms with tumor-formation in the heart, the kidneys, and the skin especially of the face, where the condition of adenoma sebaceum is developed. The prognosis is unfavorable, although a fatal ending sometimes is deferred until early adult life.

3. DIFFUSE CEREBRAL SCLEROSIS.—This is a very uncommon acquired form, beginning oftenest in infancy. Hereditary syphilis, fetal meningitis, and trauma of the head have been assigned as causes. The pathological process is a diffuse sclerosis of the base of the brain, the medulla, and the spinal cord, depending upon an overgrowth of the glial tissue. The white matter is affected more than the cortex. It has certain special clinical characteristics; principally a progressive decrease of intelligence, difficulty in speech, slowness in movement, ocular paralysis, and widespread and finally extensive rigidity with contractures. There may be nystagmus, tremor, and ataxic movements; and epileptiform convulsions may occur. The course is of short duration and the termination fatal.

4. LOBAR SCLEROSIS.—This unusual affection is congenital in origin, or develops in early life. It is probably secondary to a variety of causes, such as thrombosis or meningo-encephalitis. It may effect one or both hemispheres, with a lobar distribution. The histological changes consist in a decided increase of the glial tissue, with degeneration of the ganglion cells and nerve-fibres. The symptoms vary with the region of the brain involved, but are oftenest motor in nature, consisting of those seen in infantile cerebral palsy. The intelligence suffers also to various degrees.

5. NUCLEAR ATROPHY.—In one variety of atrophy, uncommon in early life, the process, which is degenerative, is limited to the cranial nerve-nuclei (*Nuclear Ophthalmoplegia; Nuclear Bulbar Palsy*). Some of the cases of this nuclear atrophy are merely instances of the nuclear localization of poliomyelitis. Others depend upon syphilis or the acute infectious diseases; and in some there is a familial tendency. There occurs also a *nuclear aplasia* (p. 310), which may be very similar to the nuclear atrophy in its symptoms, but differs pathologically in that it consists in a congenital failure of development of the nuclei. Cases in which the **symptoms** are due to a poliomyelitis or an aplasia do not exhibit a progressive course. On the other hand, in the more typical cases of nuclear atrophy, not dependent upon either of these conditions, the course is progressively unfavorable to a fatal ending, although often with interruptions and remissions, and only exceptionally with a permanent cessation of advance. In *progressive nuclear ophthalmoplegia* some or all of the external ocular movements are involved, ptosis being often the earliest symptom. In *progressive bulbar palsy* various nuclei in the pons and medulla are affected. There is then increasing difficulty in articulation and in swallowing; disappearance of facial expression; tremor of the tongue; inability

to retain the saliva in the mouth; palatal paralysis; hoarseness; and interference with respiration and cardiac action.

6. Disseminated Sclerosis of the Brain and Spinal Cord.—Cases of this disease have repeatedly been reported as occurring in childhood; but, although an affection of adolescence and early adult life, it would appear in fact to be uncommon at periods previous to this. Frankl-Hochwart[1] found in 206 cases diagnosed as multiple sclerosis only 8 in the first 10 years. Most frequently the disease appears to be a primary one, but it may occur as an affection secondary to the acute infectious diseases.

The **lesions** involve both brain and spinal cord, and consist of a sclerosis, or, in the primary cases, a gliosis analogous to a tumor-formation. They occur as greyish-red, firm patches, greatly varying in size, and occupying indifferently the white or the grey matter They are usually most numerous in the cerebrum, especially about the ventricles, and in the cerebellum, pons, medulla, and basal ganglia; but the cord is always involved as well, chiefly in the cervical region and the lateral tracts. Histologically there is a disappearance to a large extent of the medullary sheaths, with preservation of the axis cylinders and ganglion cells, at least until late in the disease. The special feature is a great proliferation of the neuroglia. The meninges and the blood-vessels exhibit no characteristic alterations.

The **symptoms** necessarily vary greatly, depending upon the seat of the lesions. The most characteristic are scanning speech; intention-tremor, nystagmus; bilateral or unilateral spastic paresis, particularly of the legs; and increase of the tendon-reflexes. Disturbance in the distribution of any of the cranial nerves may occur, especially in the eyes. Anesthesia or paresthesia in different regions is often seen. Some degree of mental deficiency is not uncommon.

The onset of the disease is slow and insidious; the course chronic, with remissions and acute exacerbations; yet on the whole progressive, with development from time to time of new symptoms indicating the extension of the process to new regions. The **prognosis** is unfavorable. The **diagnosis** rests upon the features detailed, but is difficult and uncertain in the absence of a post-morten examination, since any or all of the symptoms may occur in childhood and yet the disseminated lesions of this disease be absent.

HYPERTROPHY OF THE BRAIN

In the form of a true hypertrophy it is doubtful whether this affection exists. There is, however, a hypertrophic encephalitis in children, but of uncommon occurrence. It is seen in the first years of life, especially in connection with rachitis, but may be associated with other conditions, such as disorders of the thymus gland or of the adrenals, or may be congenital. The **lesions** consist especially in a hyperplasia of the neuroglia, although the nervous tissue may be involved to some extent. As the neuroglia increases the nervous elements degenerate. The cerebrum is most involved, and the total weight and size of the brain may be decidedly above normal. The convolutions are flattened and are firmer and dryer than normal, and the cerebral fluid is greatly diminished. The earliest **symptoms** consist of fretfulness, disturbance of sleep, headache, and other evidences of cerebral irritation, much as in chronic hydrocephalus. The diagnosis is rendered still more difficult by the gradually augmenting size of the head, if the condition of the sutures permits of this, the appearance being that of hydrocephalus. The **prognosis** is entirely unfavorable. As the disease advances, symptoms of increasing pressure are seen, with enfeeblement of body and of mind, a stuporous state, and finally occasional or terminal convulsions.

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[1] Arb. aus der neurol. Inst. an der Wien. Univ., 1903, X, 19.

PROGRESSIVE GENERAL PARALYSIS

(Paralytic Dementia; General Paresis)

Etiology.—Only brief reference will be made to this disease, because of its rarity in children. It is, however, more frequently encountered at this period than is another manifestation of syphilis of the nervous system: tabes dorsalis (p. 386). It is not often seen before about 12 years of age or later, and still less often before 8 or 9 years, although Zappert,[1] observed a case at the age of 5 years. Thiry[2] collected the records of 58 cases in individuals of 20 years of age or less, 18 of the patients being not over 12 years, the youngest 8 years. The number has been largely increased since then (Leonard[3] about 250 cases).

Pathological Anatomy.—The lesions consist of an atrophic condition of the whole brain, but especially of the convolutions; the result, apparently, of a diffuse meningo-encephalitis. The ventricles are dilated; a varying degree of chronic leptomeningitis is present; the connective tissue of the brain and meninges is hyperplastic; there is a syphilitic endarteritis; and the nerve cells and fibres of the cortex have more or less disappeared. The spinal cord may also exhibit lesions, chiefly in the form of degeneration of the pyramidal tracts and of the posterior columns.

Symptoms.—The symptoms are chiefly those characteristic of the disease as encountered later in life. In brief they may be divided into somatic and psychical. The former consist in a gradual increase of hesitation and indistinctness in speech; Argyll-Robertson pupil; ataxia; tremor of the lips and tongue; unsteadiness of gait; exaggeration of tendon-reflexes; increasing weakness in the limbs, and often a stunting of growth. Peculiar chewing or sucking movements constitute a common symptom in early life. There are also sudden characteristic paralytic attacks with vertigo, loss of speech, headache, local paralyses, or epileptic seizures with loss of consciousness. The psychical disturbances comprise a progressive diminution of intellectual power, with loss of interest in the surroundings, listlessness, depression, and inability to perform many acts formerly done with ease. Hallucinations and delusions are much less common than in adult life. In the later stages the mental condition is that of dementia; while the body wastes, rigidities and contractures develop, and there is paralysis of the sphincters.

The **prognosis** is entirely unfavorable, and the occurrences of temporary improvement seen in adult life are not often observed before this period. The **duration** of the disease is variable, death taking place in from a few months to several years, the average time being 3 to 4 years, the course being rather longer than in adults. Before the end is reached the symptoms of tabes may become associated, and the child exhibit loss of knee-jerks, pain and optic atrophy. The **diagnosis** rests upon the slow onset and course, the principal diagnostic symptoms being the gradual loss of mental power, the Argyll-Robertson pupils, affection of speech, and the general physical deterioration. In addition there may be other evidences of the presence of syphilis, while a Wassermann reaction may be obtainable, and the cerebrospinal fluid show a lymphocytosis.

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[1] Pfaundler und Schlossmann, Handb. der Kinderheilk., 1906, IIa, 661.

[2] Thèse de Paris, 1898. Ref., Mott, Arch. of Neurol., 1900, I, 250.

[3] Illinois Med. Journ., 1915, XXVII, 443.

INFANTILE CEREBRAL PARALYSIS

The condition designated by this title is of frequent occurrence. It is in no instance a primary disease, but is composed of the terminal group of symptoms secondary to a number of intracranial affections; varying much, yet brought together here for convenience of study. The paralyses produced by hydrocephalus, tumor, abscess, and polioencephalitis have been discussed in other sections and are not considered here. Infantile cerebral paralysis may be subdivided in various ways according to the cause, the period of life, and the distribution and nature of the paralysis. Based upon the last of these the disease resolves itself into (1) *Spastic hemiplegia*, and (2) *Spastic diplegia and paraplegia;* while following the classification of Sachs,[1] according to the time of development, one may speak of (1) *Paralysis of intra-uterine origin;* (2) *Birth* or *natal palsy;* (3) *Acute acquired paralysis*, or *postnatal palsy*.

Of these groups of cases the antenatal is less common than the other two. The symptoms here are present at birth, even although discovered only later. In the palsies produced at birth, perhaps the most frequent variety, the symptoms develop suddenly soon after this. The postnatal cases may occur at any time in early life, but the large majority begin in infancy and early childhood. With respect to the distribution of the paralysis, the prenatal cases are oftenest diplegic or paraplegic, and this is true, likewise, of the birth palsies. The acquired paralyses are much oftener hemiplegic, and almost all cases occurring after 3 years of age are of this nature. As to the relative frequency of the different forms of paralysis, Sachs[2] found in 225 cases, 156 with hemiplegia, and 69 with diplegia or paraplegia. Taking all the cases together fully 66 per cent. began in the first 3 years of life.

Etiology and Pathological Anatomy.—The lesions and causes are well shown in the following table given by Sachs.

TABLE 88.—CLASSIFICATION OF INFANTILE CEREBRAL PALSIES

Groups.	Morbid Lesion.
I. Paralyses of intra-uterine onset..................	Large cerebral defects. (Porencephaly.) Defective development of pyramidal tracts. Agenesis corticalis. (Highest nerve elements involved.)
II. Birth Palsies...........	Meningeal hemorrhage, rarely intra-cerebral hemorrhage. Later conditions: Meningo-encephalitis chronica, sclerosis, and cysts; partial atrophies.
III. Acute (acquired) Palsies.	Hemorrhage (meningeal, and rarely intra-cerebral); thrombosis (from syphilitic endarteritis and in marantic conditions); embolism. Later conditions: Atrophy, cysts, and sclerosis (diffuse and lobar). Meningitis chronica. Hydrocephlaus (seldom the sole cause). Primary encephalitis; polio-encephalitis acuta (Strümpell).

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[1] Nervous Diseases of Childhood, 1905, 447.
[2] *Loc. cit.*, 433.

1. Paralysis of Intra-uterine Origin.—The condition of the mother before parturition may have some bearing; and it is very possible that injuries received by her, such as blows upon the abdomen, may produce damage to the brain of the fetus. Among the lesions of large size are cysts, microcephalus, and porencephalus of various forms (see p. 309); which may be the result of hemorrhage, thrombosis, meningo-encephalitis, or defective development. An imperfect formation of the cells of the cortex, especially the pyramidal cells (Agenesis Corticalis, see p. 310), is an occasional cause. Of the same nature is the imperfect structure of the brain present in prematurely born children, which is normal for the age to which they had attained at birth, but not suitable for an infant after birth. This readily gives rise to spasmodic and paralytic symptoms which, although appearing at or shortly after birth, are in reality dependent upon conditions belonging to intra-uterine life.

2. Birth Palsies.—The primary factor in nearly all such cases is meningeal hemorrhage, the result of trauma incurred during birth. (See Vol. I, p. 265; Vol. II, p. 314.) This is oftenest the result of long-continued, difficult labor. In other less frequent instances the hemorrhage would appear to be produced by a too hurried birth, with the great and rapidly changing internal pressure which results. The effusion of blood is oftenest from the pial vessels; sometimes from the longitudinal sinus. It is most frequently vertical in breech presentations, and basilar in vertex presentations; but to this there are exceptions. The various lesions found secondary to the meningeal hemorrhage are shown in the table as given, and have been described under Meningeal Hemorrhage (p. 314). In about 25 per cent. of the cases the spinal cord is affected also.

3. Post-natal Palsies (*Acquired Palsies*).—A large number of pathological lesions have been found (see table), many of which are only terminal processes. The primary lesions are most frequently hemorrhage, thrombosis, embolism, and encephalitis; the latter dependent upon gastrointestinal disorders or upon some of the infectious diseases, including the cerebral form of poliomyelitis. The method of production in these cases has been discussed in the separate chapters treating of them. Of these meningeal hemorrhage is much the most frequent; intracerebral hemorrhage being rare. Embolism also is an exceptional etiological factor, but thrombosis of the vessels and sinuses is more frequently seen. Diffuse sclerosis of the brain is an unusual cause (p. 362), and very probably hereditary syphilis may be an agent through the production of a chronic syphilitic meningitis.

Symptoms.—For convenience of clinical study the cases may be divided into (*A*) hemiplegia; (*B*) diplegia and paraplegia. Pure monoplegia is so rare that it may be disregarded.

(*A*) INFANTILE HEMIPLEGIA.—The term infantile is used here in a broader sense, including infancy and childhood, and is applicable on account of the great preponderance of occurrence during the first of these periods. In the majority the disease is of the acquired variety, and seen oftenest from a few months up to 3 years of age. In 80 cases studied by Gowers,[1] not including any cases developing at birth, 60 per cent. developed in the first 2 years of life, and about 88 per cent. in the first 5 years. Comparatively few hemiplegias are of the natal or prenatal class. As regards the relative frequency of hemiplegia and diplegia, Sachs and Peterson[2] found 105 hemiplegias in 140 cases of cerebral paralysis; while

[1] Dis. of the Nerv. Syst., 1888, Amer. edit., 840.

[2] Jour. Nerv. and Ment. Dis., 1890, IV, 295.

in Osler's[1] 151 cases there were 120 of hemiplegia. In both these series the studies were made for the most part on subjects past the periods of infancy and early childhood; and since most of the diplegias are short-lived, the incidence of this latter form of cerebral paralysis is certainly greater than these figures indicate. In fact, it is probable that the majority of the cases occurring in infancy are instances of diplegia (Holt and Howland).[2]

The onset of hemiplegia is usually sudden, the first symptom being often a prolonged convulsive attack, most marked on, or confined to, one side of the body. This may sometimes last with interruptions over several days. Convulsions marked the onset in 52 of 97 cases collected by Osler,[3] and in 43 of 88 cases reported by Wallenberg.[4] The temperature may be elevated; the consciousness is lost between the convulsive seizures if the case is severe. Paralysis of a hemiplegic nature, including the face on the affected side, appears early, but often may be overlooked on account of the gravity of the general condition. In infancy and early childhood speech is frequently lost at first, if it has previously existed, no matter which side of the brain is involved. It may, too, be rendered difficult or indistinct by the facial paralysis. In later childhood in right-handed subjects speech is affected only when the lesion is in the left hemisphere. As time passes the paralysis becomes of a decidedly spastic type with rigidity, with increase of the tendon-reflexes and with ankle-clonus of the affected side if the stiffness permits of obtaining these, and with the presence of the Babinski[5] toe-reflex, although this is of value only after the age of 3 years. The rigidity may vary in degree in different cases, and in the same case from time to time. There are occasional instances, however, in which there is a flaccid condition of the body present instead of rigidity. As improvement takes place the lower extremities and the face recover usually more quickly and to a greater extent than the arm, but generally some degree of involvement of the leg remains, producing a limp. Sometimes, however, one limb, generally the leg, may recover so completely that the condition as seen later appears to be monoplegia. On the other hand, many cases do not exhibit such a degree of improvement of the leg, but a typical spastic paralysis remains with equinovarus, stiff dragging with outward swinging of the extremity, and a gait upon the ball of the toes; or the limb may be too weak to permit of standing upon it alone (Fig. 351). In some cases, contrary to the rule, the leg remains decidedly affected and the arm very little so. The degree of residual paralysis and of spasticity, respectively, in the

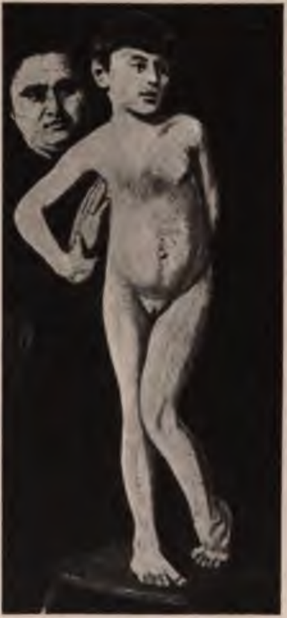

Fig. 351.—Infantile Hemiplegia, Later Stage. (*Sachs, Nervous Diseases in Children*, 1895, 291.)

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[1] Cereb. Palsies of Child., 1886, 4.

[2] Dis. of Infancy and Childhood, 1916, 781.

[3] *Loc. cit.*, 26.

[4] Contrib. a. l'étude de hemiplegie cérébrale infantile, 1884. Ref., Osler, *loc. cit.*, 20.

[5] Sem. méd., 1898, XVIII, 321.

affected extremities varies with the case. Sometimes paresis predominates, in other cases rigidity. Involvement of cranial nerves other than the facial is only occasionally observed.

Later in the course there persists a moderate degree of atrophy with shortening of the limb; scoliosis; and asymmetry of the pelvis, the shoulder-girdle, and sometimes of the face, resulting in a facial hemiatrophy. The atrophy of the limbs is not of the degree or of the flaccid type seen in poliomyelitis. The general bodily development is often much below normal. Contractures may appear after the lapse of 1 or 2 years, or occasionally soon after the onset, producing to a varying degree talipes in the foot, flexing of the wrist and elbow, pronation of the forearm, and adduction of the upper arm to the side.

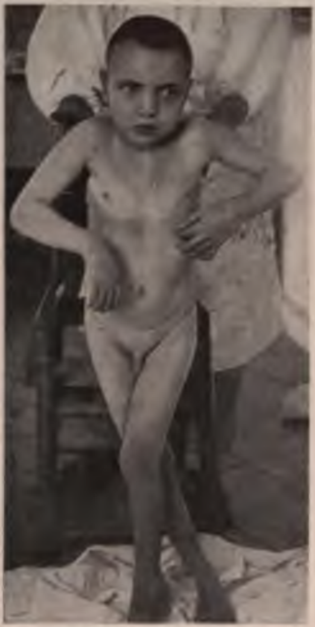
FIG. 352.—SPASTIC DIPLEGIA.

Illustrating cross-legged progression. (*Concetti, Arch. f. Kinderh.*, 1913, *LX–LXI*, 175.)

Sensory disturbances are rarely marked and are usually absent, and there is no alteration of the electrical reactions, other than a moderate quantitative diminution of response. Among later disturbances of a post-paralytic nature, coming on from 9 to 24 months after the onset, may be mentioned athetosis and choreiform movements of various sorts, most marked on voluntary effort. Unilateral tremor of the paralyzed limbs is sometimes observed but is not of frequent occurrence. Epilepsy is a frequent sequel. It may sometimes be of the Jacksonian type, but may later become general in character. Some degree of mental defect is frequently but not necessarily present, and is not so often witnessed as in diplegia; many subjects remaining entirely normal mentally. The mental defect is the result of a diffuse cerebral sclerosis which has spread from the original focal lesion. It is oftenest seen when the frontal lobes are involved.

(*B*) SPASTIC DIPLEGIA AND PARAPLEGIA. LITTLE'S DISEASE.—Diplegia and paraplegia, both dependent upon a bilateral intracranial lesion, differ from each other chiefly in the limitation of the paralysis in the latter to the lower extremities. Nearly all of these cases are diplegic at the beginning, and although the paralysis in the arms later recovers completely and only paraplegia remains, other symptoms are still present, which show the early nature of the case. Nearly all, too, are prenatal in origin or occur at birth. Only rarely are they of the acquired type. The symptoms of paralysis are in diplegia much less prominent than those of other nature. The lower extremities are often much more involved than the upper. The face is frequently attacked at first, but later appears to be normal. The two sides of the body are seldom affected equally. Generally rigidity is one of the most prominent manifestations, either present at birth or developing soon after it, although none of the symptoms may have been observed by the parents until the time comes for sitting or walking. Contractures soon appear in the majority of cases. The degree of stiffness of the whole body is often remarkable, and there may be opisthotonos; and, as the child grows older, voluntary efforts are, in severe cases, almost impossible on account of the incoördination present. A child may be unable for some years to sit unsupported or to hold its head up. Walking is impossible, or, if accomplished unaided or with assistance, exhibits a typical cross-legged progression due to the adductor spasm (Fig. 352). Increase of the tendon-reflexes is marked; ankle-clonus is present; and bilateral athetosis or choreiform movements are frequently seen. In other instances inability or slowness in attaining the power to sit or hold the head erect may be dependent rather on the general physical and mental debility.

In occasional instances the spastic condition is replaced by one of hypotonia. This "atonic astasic" type of diplegia, as denominated by Förster,[1] or hypotonic form, is characterized by a general muscular hypotonia and inability to stand, sit, or hold the head erect; although the child while lying in bed can make ordinary movements with the limbs, and the muscle-strength appears to be preserved. The tendon-reflexes and electrical reactions are normal. With this is associated very decided mental defect. The lesion is supposed to be situated in the frontal lobes. The condition has been further described by Clark,[2] Batten and von Wyss,[3] and others.

The incoördination present in cerebral diplegia may make speech difficult or impossible; or the lack of speech or the slow acquiring of it may depend upon the mental defect which is so common in this type of paralysis, and which ranges from a slight impairment up to complete idiocy. As regards the relationship of the speech to the mental condition, although a defective intelligence is the most frequent cause of defective speech, I have seen cases of the most spastic incoördination in which, as far as could be determined, the intelligence was unaffected, but talking was entirely impossible on account of the lack of muscular control. Involvement of the cerebral nerves may be present, especially that producing optic atrophy, and there may occasionally be an *infantile pseudobulbar palsy* suggesting bulbar paralysis, yet dependent upon lesions of the cortex; not of the pons and medulla. The symptoms of this consist especially in difficulty in swallowing, interference with speech, hanging of the jaw, dribbling of saliva, immobility of the tongue, and inability to chew. Epileptiform convulsions are observed in fully 50 per cent. of the cases of diplegia. They may appear early, or sometimes at frequent intervals later. The head may be variously malformed, or unduly small, the degree depending upon the extent of the involvement of the brain. The paralyzed limbs are smaller than normal.

Not all cases show such marked symptoms; and in the milder ones there may be only a certain weakness of the muscles, increased knee-jerks, some rigidity of the lower extremities, or an occasional stiffness of the whole body; with more or less mental defect which shows itself as time passes, although occasionally the intellectual power is normal. The symptoms in these milder cases may not become apparent until a number of months after birth.

Little's Disease.—The term Little's Disease[4] has been applied broadly to all forms of cerebral spastic diplegia. It would seem to be more properly limited to those cases observed in prematurely born or imperfectly developed infants, in which there existed in consequence of this a defective formation of the pyramidal tracts in the brain and cord. A spastic rigidity dating from birth, with some degree of loss of power, is the most characteristic feature. This involves, to any considerable degree, only the lower extremities, the arms remaining entirely or quite free. The speech is not necessarily affected, and convulsions are uncommon. The intelligence may be entirely normal or slightly defective. Improvement in all the symptoms may proceed to a certain extent as time passes, although some degree of spastic condition remains.

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[1] Deut. Arch. klin. Med., 1909, CXVIII, 216.

[2] Amer. Jour. Dis. Child., 1913, V, 425.

[3] Brit. Jour. Dis. Child., 1915, XII, 65.

[4] Lancet, 1843, I, 318.

Cerebral Monoplegia.—This is, as stated, so uncommon that it is of little clinical importance. It is, however, to be remembered that it is possible for a brachial monoplegia of cerebral origin to occur, which could be confounded with the brachial injury resulting in "obstetrical paralysis." (See p. 404.)

Course and Prognosis.—The prognosis of infantile *hemiplegia* is, on the whole, good as regards life; that for complete recovery is unfavorable. The degree of improvement depends upon the severity, nature, and extent of the lesions, and the age of the patient at the time of the onset. The older the child when the disease occurs the less likely, as a rule, is serious damage to the brain to have taken place. The more marked and extensive the early symptoms, the more uncertain is the final outcome. Yet this is true only to a certain degree, and it is impossible to determine at the beginning of the attack, particularly in infancy, how complete the recovery will be, and especially what will be the final mental condition. Most children will certainly show some traces of paralysis, spastic state, deformity, or some psychic disturbance remaining, although this may in favorable cases be very slight and readily overlooked. The paralytic symptoms are less likely to persist than the spastic. A very decided permanent paralysis may be accompanied by perfect mental power; and, on the other hand, mental deficiency or epilepsy, or both, may remain the most prominent final symptom. About 66 per cent. of Gowers'[1] 80 cases of cerebral paralysis developed epilepsy. It was seen, too, in 76.4 per cent. of Koenig's[2] series of cerebral paralysis in children, and in 40 per cent. of Fuch's[2] 100 cases. Defective intelligence of some degree is very likely to remain in the hemiplegic cases. As regards the persistence of this, 80 of Sachs and Peterson's 140 cases (57.1 per cent.) of cerebral paralysis had some degree of mental impairment, severe or slight, and Ziehen[3] believed that decidedly over 50 per cent. of the hemiplegic cases suffered in this way. Those with diplegia and paraplegia are still more likely to experience some mental impairment. Thus in an analysis of 225 cases made by Sachs[4] idiocy was present in 35 per cent. of the cases of diplegia, 60 per cent. of the paraplegias and 13 per cent. of the hemiplegias.

The rapidity with which convalescence begins and advances is likewise variable. A prognosis can be made only after several weeks have elapsed after the onset, by which time it may have become evident that no improvement will occur; or in the favorable cases recovery then commences, first and most marked in the leg in most instances, while the power of speech begins to return, unless the mind has been affected. A persistent aphasia was observed in only 17 of Sachs and Peterson's

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[1] Nerves Diseasses of Childhood, 1905,843.

[2] Ref. Bruns, Cramer u. Ziehen, Handb. d. Nervenkr. im Kindersalter, 1912, 636.

[3] Bruns, Cramer and Ziehen, Handb. d. Nervenkr. im Kindersalter, 1912, 635.

[4] Nervous Diseases of Childhood, 1905, 445.

105 cases of hemiplegia. Persistence of facial paralysis was seen in 22 of their 140 cases of cerebral paralysis. The repeated occurrence of convulsions, as improvement in other respects goes on, makes one fear that symptomatic epilepsy is developing. It not infrequently happens, however, that epilepsy does not appear for even a year or more after the original lesion.

The prognosis in all the *diplegic* cases—nearly all of which, as stated, are natal or prenatal in origin—is very uncertain even as regards life. Those in which there are repeated convulsions in the early weeks have probably a severe lesion. Many cases die during the acute symptoms, and others succumb later in infancy, often from inanition. It is impossible for several months to predict what the outcome will be as regards the persistence and nature of the final symptoms. A very large proportion of those who survive are left with permanent mental weakness or epilepsy; and many will retain a degree of rigidity of the limbs and body which makes them physically helpless. In general, the absence after a few months of marked spasticity and of repeated convulsions, and the return of some power of the limbs, makes the prognosis more favorable.

An exception to the unfavorable prognosis of diplegia exists in the case of *Little's disease*, in which there is a tendency to complete recovery of mental power, and to a steady improvement to a certain extent in the control of the muscles.

Diagnosis.—The diagnosis of well-marked infantile cerebral paralysis should usually offer no difficulty. There is the combination of paralysis with rigidity, increased reflexes, contractures, and frequently mental deficiency; together with absence of marked atrophy, sensory disturbance, and alteration of electrical reactions. In spite of this, mistakes are easily and often made. One cause of error is the failure of the physician to remember that, especially in the diplegic cases, paralysis is often the least prominent symptom; the spasm, incoördinate movements, or deficiency of intellect occupying the foreground. Then, too, in the mild hemiplegic cases, the spastic rigidity may be slight and readily overlooked; or the paralysis may appear at first sight to be monoplegic, and the diagnosis of a lesion outside of the brain is consequently made. Careful study will generally settle the question. It may be that epileptic attacks occur conjointly with the other symptoms and point conclusively to a lesion of the brain; or an exaggerated knee-jerk upon one side may be the only remainder of a paralysis of the leg, and may thus clear the nature of what had seemed to be a brachial monoplegia of other than cerebral origin.

Among the conditions often confounded with cerebral paralysis is that due to *poliomyelitis*. The atrophy in this affection is, however, much greater; the reflexes diminished or absent; there are altered electrical reactions; the paralysis is flaccid; and the distribution of this is not hemiplegic or diplegic, but markedly greater in certain limbs than in others. The paralysis of *Pott's disease*, although spastic in type, has a history entirely different from that of cerebral paralysis. *Obstetrical paralysis* of one arm is due to damage to the nerves during birth, and exhibits a flaccid paralysis with atrophy. Its monoplegic character is also decisive. The pseudoparalysis of some cases of rickets suggests rather a poliomyelitis. Occasionally the spastic condition in diplegia, especially of the hands and feet, has been supposed to be due to *tetany* of the persistent type. I have seen this mistake made. The history, however, should be sufficient to differentiate them, and there is little real resemblance. *Pontine tumors* may produce spastic symptoms suggesting cerebral diplegia, but the presence of the usual evidences of intracranial tumor, combined with paralysis of some of the cranial nerves, will generally serve to distinguish. The form of infantile cerebral paralysis with especial involvement of the cranial nerves, designated *pseudobulbar palsy,* is distinguished from that in which the nuclei of the medulla and pons are atrophied (p. 362), by lack of progressive increase of symptoms, trembling of tongue, and atrophy; while the cardiac action and respiration are seldom affected. Cases of congenital *nuclear aplasia* (p. 310) might perhaps be included as a form of congenital infantile diplegia. Postparalytic chorea is distinguished by its history and by its persistence. Postparalytic epilepsy is recognized by the history of the case and the existence of evidences of infantile paralysis. (See Epilepsy, p. 242.)

Treatment.—The treatment in the early stages is that suitable for the cause, so far as this is possible. After the quiescent stage is reached the only treatment which can be employed is symptomatic, and that tending to prevent deformity, strengthen the muscles, lessen the rigidity, and improve the mental condition. It is not very satisfactory. Hot baths and passive movements with over-extension and over-flexion relieve the spastic condition to a certain degree; while massage may be used for the same purpose, and to improve the strength of the muscles. Orthopedic apparatus is often required to prevent deformities, and tenotomy to relieve contractions of long duration. For the nervous irritability of those with mental deficiency, epileptic convulsions, and rigidity, the bromides or other sedatives may be given. The treatment of choreiform and athetoid movements is not very successful. Careful training may do much for the improvement of mental deficiency, if of moderate degree. Tendon-transplantation, nerve-transplantation, nerve-stretching, and division of the posterior nerve-roots, are operative procedures which have been attempted with occasional benefit.

CHAPTER VI

DISEASES OF THE SPINAL CORD

Diseases of the spinal cord may occur as primary affections limited to this region; may be secondary to disorders of the brain, such as tumor and hemorrhage; or may be part of a process which involves simultaneously both brain and cord or their membranes; such, for instance, as poliomyelitis, cerebrospinal fever, and disseminated sclerosis. There can, therefore, be no sharp demarcation in the classification of the disorders of the two regions. The following descriptions apply for the most part especially to the conditions where the cord alone is affected, or the most marked symptoms are referable to disease there.

MALFORMATIONS

The variety of these is not great. They are frequently found in combination with malformations of the brain or of other parts of the body. Most of them are of very exceptional occurrence, much the most frequent and important being spina bifida.

SPINA BIFIDA

Etiology and Pathological Anatomy.—This lesion is analogous to cerebral meningocele and encephalocele, but is much more frequently encountered and more important. Chaussier[1] recorded it 22 times in 22,293 births; *i.e.* about 0.1 per cent., and Demme[2] reported on 57 cases in 36,148 sick children, a ratio of 1 : 634. The cause is obscure. There is sometimes a familial tendency observed. It is often associated with other deformities, such as hydrocephalus, club-foot, hydromyelia, hare-lip, cerebral encephalocele or meningocele, ectopia of the bladder, congenital hernia, and the like (Fig. 348). It depends upon a failure of development occurring early in embryonic life, as a result of which the bony arches of the spinal canal do not completely close. The opening may be small, and no effusion of cerebrospinal fluid or projection of membranes take place (*spina bifida occulta*). The lesion in this form is usually situated in the lumbar region. In the more typical cases there is a yielding of the soft tissues, as well as the separation of the bony arches, and commonly a perforation of the dura, and a tumor is produced, globular in shape, tense, elastic, fluctuating, and of a size which may equal several inches in diameter. In the simplest and most unusual form of this only the membranes protrude (*meningocele*), the fluid which distends them being either in the arachnoid cavity or the posterior subarachnoid space. The opening connecting the sac with the spinal canal is very small or may be closed. This variety is situated oftenest in the cervical or sacral region. It is globular, transparent, often pedunculated, and covered with skin of normal appearance.

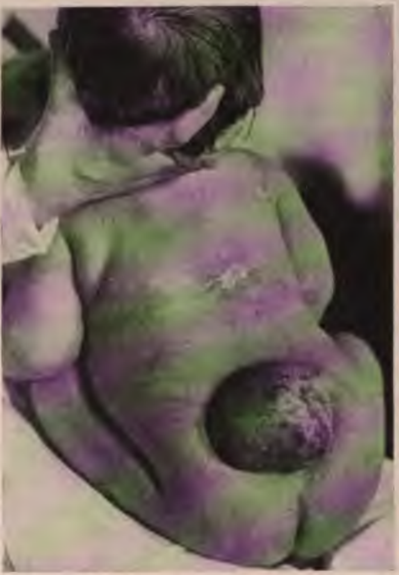

FIG. 353.—SPINA BIFIDA.
Courtesy of Dr. H. R. Wharton.

Much the commonest and the most serious form is *meningomyelocele*. Here both the spinal cord and the meninges are contained in the sac, which is usually smaller than in the previous variety. The tumor is oval, soft, fluctuating, sessile; the skin over it is reddish and vascular and usually depressed at the centre, where it is very thin and frequently ulcerated (Fig. 353). The spinal cord forms a part of the wall of the sac. The fluid accumulates in the anterior subarachnoid space or anterior arachnoid cavity. This form of spina bifida is oftenest found in the sacrolumbar region; sometimes in the cervical.

The *myelo-cystocele* (syringomyelocele; hydromyelocele) is an uncommon variety, in which the central canal of the cord dilates and pushes the porterior portion of this with its coverings through the opening in the spinal canal. This produces a tumor, sessile on a broad base, and

[1] Ref., Fürst, Gerhardt's Handb. d. Kinderkr., 1880, V, 1, 1, 347.
[2] Wien. med. Blätter, 1884, VII, 804.

situated oftenest in the dorsal, dorsolumbar, or sacral region. On palpation it is found to contain solid contents as well as fluid. The skin covering it is at first normal, but may later become thin and ulcerate.

Symptoms.—These vary with the form of spina bifida present, as well as with the situation. The *spina bifida occulta* may show only a slight depression in the skin, often covered by a hairy mole, or there may be nothing abnormal visible. Adhesions of the cord to the skin may produce interference with its growth, resulting in incontinence of urine, paralysis, and neuralgias, although the development of these symptoms may be deferred until puberty. *Meningocele* is unattended by any symptoms of paralysis; and this is often true of *myelocystocele*, while in other cases of this latter form paralysis is present. *Meningomyelocele* exhibits distinct symptoms. There are often decided evidences of paralysis, their nature depending upon the position of the malformation. Frequently these consist in flaccid atrophic paralysis of the legs, more or less complete, and of the bladder and rectum; sometimes prolapse of the rectum and of the uterus; flexion of the thighs at the hip-joint; anesthesia; and trophic disturbances, such as ulceration of the buttocks, genital region, and inner surface of the thighs. Less severe cases, especially if the malformation is in the sacral region, may present only incomplete paralysis of the legs and feet, and perhaps of the sphincters as well.

Course and Prognosis.—There is always a tendency for the lesion to increase in size, and the occult form may finally develop a visible tumor when adolescence is reached. In spina bifida of other forms the tumor is present at birth and may grow rapidly. The ulcerated skin in meningomyelocele may finally become covered by epithelium, if the patient lives long enough; but septic infection is very liable to occur, with secondary involvement of the spinal and cerebral meninges. Sometimes the infection is local in the sac only, and in this way a cure may be wrought. There is always danger of rupture occurring, with a rapidly fatal ending from convulsions or from infection following the rupture. Spinal meningocele grows to large size and seldom ruptures or becomes infected.

The prognosis is on the whole unfavorable. Meningocele interferes least seriously with the patient's general condition, is more amenable to operative treatment, and the subjects may attain adult life; while meningomyelocele, on the other hand, is very liable to terminate fatally within a few weeks. The prognosis of spina bifida is especially bad when there is well-marked paralysis, and still more so if a complicating hydrocephalus exists—an association present in 17 of Demme's 57 cases. If not dying from infection or from rupture, the patient succumbs soon to inanition or diarrhea. Without operation the mortality is very high. Of Demme's series, 25 were operated upon with 7 recoveries. Of the 32 cases not operated upon, all died, and 25 of these by the end of the 1st month. Even with operative interference the majority do not recover, and in those in which it has been successful hydrocephalus may develop later.

Diagnosis.—The diagnosis is usually readily made; based upon the appearance of the tumor and the associated symptoms. It is only in the absence of such symptoms that such lumbosacral tumors as lipoma and teratoma can cause any confusion. These have not, however, the fluctuating character of spina bifida. The recognition of the different varieties of the disease is often extremely difficult. Translucency and fluctuation with normal cutaneous covering, and the absence of a spinal fissure which can be discovered by palpation, point to meningocele. The broad base and the very thin-walled, red, depressed central portion of the sac, together with paralytic symptoms, make a meningomyelocele probable. A broad-based tumor with normal cutaneous covering indicates myelocystocele, especially if associated with hydrocephalus and without paralytic symptoms. The recognition of spina bifida occulta is often impossible. It may be suspected when there exists paralysis and neuralgic pains in the lower extremities, and weakness of the sphincters; but these symptoms may readily be produced by other lesions.

Treatment.—Surgical treatment is that indicated for suitable cases. Various methods have been employed, among them injection into the sac, ligation, compression, and the like. These have largely given place to plastic operations of different sorts. Surgical intervention is, however, without benefit and is contra-indicated when hydrocephalus is also present; when the covering of the sac is of such a nature that flaps cannot be formed; or when there are attendant severe paralytic symptoms. The operation is in itself dangerous, and even after apparent surgical recovery the symptoms may remain unchanged, or hydrocephalus develop later. The fact that a portion of the spinal cord is present in the sac in the large majority of cases explains the frequent failure to obtain benefit from surgical treatment. Meningocele offers the best hope, and the operation is also justified in myelocystocele, or in other cases where rupture is threatening. The occult cases may frequently be successfully operated upon with relief of the symptoms. In the absence of operation all that can be done for spina bifida is to employ antiseptic applications to prevent infection, and to guard against rupture.

OTHER MALFORMATIONS OF THE SPINAL CORD

These are of rare occurrence and of little practical importance. There may be complete absence of the spinal cord (**amyelia**), or entire failure of closure of the spinal canal posteriorly (**rachischisis**), with splitting open of the cord—conditions incompatible with life. In other cases there is partial deficiency of the cord (**atelomyelia**); **heterotopia** of small areas of grey matter into the white matter; partial **degeneration** of the pyramidal tracts; a congenital dilatation of the central canal (**hydromyelia**); or a doubling of the cord (**diplomyelia**) or of the central spinal canal (**diastematomyelia**) through a portion of its extent. With all of these conditions malformations of other regions of the body are very prone to be associated. To hydromyelia there may be later joined a **syringomyelia,** dependent upon a diffuse gliosis of the cord, with the formation of cavities. It seems doubtful whether syringomyelia ever occurs alone in early life.

SPINAL MENINGITIS

As in the case of the brain, inflammation may affect the dura mater (*spinal pachymeningitis*), or the pia and arachnoid (*spinal leptomeningitis*), or the two may be combined. The inflammation may be acute or chronic, primary or secondary.

1. PACHYMENINGITIS.—As a primary disease this probably does not occur. The most frequent cause is extension from spinal caries; less often it is secondary to tumor or to trauma; and sometimes it depends upon suppuration outside of the spine, as in retropharyngeal abscess. In most instances the inflammation is limited to the external surface of the dura mater, and is of a semipurulent character, termi-

nating in an abscess with cheesy pus. Symptoms develop, the result of the compression myelitis which is produced and of the pressure upon the nerve roots. (See Compression Myelitis, p. 380.) The early symptoms differ from myelitis in the slower onset, the preponderance of pain and muscular spasm, and the absence of paralysis.

Pachymeningitis cervicalis hypertrophica is a rare affection in which a firm deposit of fibrous tissue develops on the inner layer of the dura mater, as in the case of cerebral internal pachymeningitis with which it may be associated. The symptoms resulting are those of compression myelitis of the cervical region with involvement of the nerve roots; the most prominent being pain in the upper extremities, shoulder, and neck; hyperesthesia; and atrophy, partial paralysis, and contractions of the hands. The onset is gradual, and the course slow, lasting for years. The prognosis is serious and the case may terminate fatally; but, on the other hand, arrest of the process may occur, or recovery may be complete.

2. SPINAL LEPTOMENINGITIS. **Etiology.**—The disease may be acute or chronic, and is usually combined with the analogous inflammation of the brain seen in cases of cerebrospinal fever or of tuberculous meningitis. In the former the lesions may be widespread, or chiefly dorsal and lumbar; in the latter oftener confined to the cervical region of the cord. It is possible, too, that a gummatous meningitis may involve the dura and meninges of the brain and cord. As a localized process spinal meningitis may also depend upon spinal caries, tumor, and trauma. The inflammation may readily extend to the cord itself, producing a meningomyelitis.

Symptoms.—The principal symptoms present are usually those occurring in connection with the form of cerebral meningitis with which the spinal inflammation may be combined. In cases where the spinal involvement predominates, or where the meningitis is localized only in the spine, pain becomes the most prominent early symptom, accompanied by a moderate febrile reaction. The pain is more or less paroxysmal, very severe, worse on movement, and felt in the back and limbs. There may be paresthesia, hyperesthesia, or hyperalgesia. Rigidity, contractures, painful spasmodic contraction of the extremities, increased reflexes, stiffness of the neck, or opisthotonos may appear. Paralytic manifestations succeed these symptoms, relaxation replacing rigidity, sensation being lost, and paralysis of the sphincters perhaps being present. The nature and position of the symptoms depend largely upon the situation of the lesion. If the lumbar cord is affected, the lower extremities suffer; if the dorsal, there is added to this involvement of the trunk as well; if the cervical region, there may be difficulty in respiration and disturbance of the movements of the arms. Such symptoms as Cheyne-Stoke's respiration, coma, and delirium develop only when the cerebral meninges share in the process.

Prognosis.—The prognosis is uncertain. Omitting cerebrospinal fever and the combined tuberculous meningitis of the brain and cord, neither of which are considered here, the probability of recovery depends upon the extent and severity of the inflammation, its cause, its nearness to the medulla, and the degree to which the cord itself is involved. Some acute cases die in a few days. Cases of a chronic nature are liable to terminate fatally after long-continued exhaustion attended by muscular atrophy. Spinal meningitis depending upon trauma gives a better prognosis than that due to other causes.

Diagnosis.—The diagnosis rests chiefly upon the symptoms of meningeal irritation as described, viz. pain, rigidity, and hyperesthesia. Myelitis gives little pain; rheumatism causes pain only on movement; tetanus produces rigidity without early fever. Subdural hemorrhage is of very sudden onset, and develops the symptoms of acute meningitis a short time later.

Treatment.—For any form of spinal meningitis the treatment is chiefly that of the cause. The administration of mercury and of the iodides may be tried in cases suspected of being syphilitic in origin, and even in others. Caries of the spine must be handled along the lines appropriate for this; and trauma have its results removed as far as possible. In addition, acute spinal meningitis may be treated by an ice-bag to the spine or the application of dry cups. Remedies to relieve the pain are often necessary.

HEMORRHAGE INTO THE SPINAL MENINGES (HEMATORACHIS) OR INTO THE SPINAL CORD (HEMATOMYELIA)

Etiology.—From a clinical standpoint both of these conditions are of rare occurrence, although they are not infrequently found at autopsies upon new-born children, especially after difficult or prolonged labor. I have seen 3 cases, one confirmed by autopsy, following violent efforts at extraction in breech presentations. They are then liable to be associated with hemorrhage into other parts of the body, oftenest within the cranium. Among very exceptional causes are trauma of other nature, rupture of vessels in the course of pertussis, and hemorrhagic diseases. The hemorrhage may be extradural, subdural, into the substance of the cord, into the central canal, or in more than one of these regions; and its size may vary from punctate to large focal accumulations in the cord substance; or from a small amount to a large effusion in the case of meningeal hemorrhage, perhaps filling most of the arachnoid cavity.

Symptoms and Diagnosis.—*Meningeal hemorrhage* produces definite and characteristic symptoms, depending largely upon the seat of the lesion and upon the amount of bleeding. These consist of severe radiating pain, hyperesthesia or paresthesia, and muscular rigidity; while later there are paralysis of the limbs or sphincters and diminished sensation in the parts below the seat of the hemorrhage. The symptoms are similar to those of spinal meningitis, but hemorrhage is distinguished by the very sudden onset. In *hemorrhage into the substance of the cord,* unless of the slowly infiltrating form, the onset is equally sudden but paralysis is the most prominent early symptom, and the evidence of involvement of the meninges and of the nerve roots is absent or less marked. There are also varying focal symptoms of a nature subject to the position of the hemorrhage. In some cases of intraspinal hemorrhage, as in the 3 referred to, there is loss of tendon-reflexes and of sensation, with complete paralysis of the flaccid type. This hypotonic state may occur whether or not the lumbar enlargement of the cord is involved; and in the latter event is believed by Gött[1] to be dependent upon injury to the nerve roots through secondary meningitic thickening. A primary myelitis or meningitis has rise of temperature among the early symptoms.

Course and Prognosis.—The prognosis is always serious. Meningeal hemorrhage is liable to be followed by death as paralytic symptoms develop, and this is especially true if the effusion is in the meninges of the upper part of the cord. Yet partial or even complete recovery sometimes occurs if the patient has passed safely through the first 2 weeks. In hematomyelia the prognosis for recovery is even more doubtful, since secondary myelitis is likely to develop and permanent damage to remain even if the patient survive.

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[1] Jahrb. f. Kinderh., 1909, LXIX, 422.

Treatment.—The first principle of treatment is to prevent further hemorrhage, if possible, by the use of absolute rest upon the face or the side; the application of ice-bags to the seat of the lesion, and the administration of purgatives.

TUMORS OF THE SPINAL CORD

Tumors of the spinal cord are of very uncommon occurrence in children. They may be either primary or secondary, and involve either the meninges or the cord itself. In the former situation they are oftenest sarcoma or gliosarcoma; in the latter tubercle or sometimes glioma. Gummatous tumors are uncommon. In 50 cases collected by Mills and Lloyd[1] 4 were in subjects under 10 years and 3 in those from 10 to 20 years; and in Schlesinger's[2] extensive series of 251 collected cases there were 33 from 1 to 9 years, and 27 from 10 to 19 years.

The **symptoms** are very varied, and there may even be none whatever. One of the most prominent and earliest is pain in some of the extremities, produced by the irritation of the nerve roots. Sometimes there is hyperesthesia also, or spastic rigidity and contractures, and later there may be loss of sensation or of motion. The character and location of the symptoms depend upon the extent and position of the lesion. When the meninges are chiefly involved evidences of irritation predominate; when the tumor is primary in the cord itself loss of motion or of sensation occupies the foreground. A growth in one side of the cord may produce a Brown-Sequard paralysis; while if the whole transverse section is involved the symptoms suggest those of transverse myelitis.

The onset and course of the disease are usually slow and may suggest caries of the spine. The **prognosis** is very unfavorable except in cases where the diagnosis can be made with sufficient certainty to permit of operation; and such diagnosis is rarely possible in early life.

SYRINGOMYELIA

Reference has already been made to this (p. 375) as a condition extremely rare in early life, so far as clinical manifestations are concerned. It is probably in most instances a gliomatous infiltration of the spinal cord, with the production later of cavities, and may consequently be considered among the new growths. The disorder may be combined with dilatation of the central spinal canal (p. 375). The infiltration and subsequent cavity-formation begin near the central spinal canal and may involve the commissure, the anterior and posterior horns, and the posterior columns. The cavities may be small and limited to one region, oftenest the cervical, or may extend throughout much of the length of the cord.

The course is very slow, and even although the pathological process very probably begins in infancy, or is even congenital, symptoms are rarely observed during childhood. The subject as far as childhood is concerned, has been carefully reviewed by Bruns.[1] The symptoms are those seen in adult life: viz. muscular atrophy and paralysis of one limb, or of the corresponding one on the other side also; various vasomotor and trophic disorders; and characteristic sensory disturbances, chiefly loss of pain-sense and temperature-sense but with preservation of tactile sensibility.

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[1] Pepper's Syst. of Med., 1886, V, 1090.

[2] Beiträge z. Klinik der Rückenmarks und Wirbeltumoren, 1898, 99.

MYELITIS

This term may be applied broadly to a variety of affections in all of which inflammation of more or less of the spinal cord is present. Among these are poliomyelitis, amyotrophic lateral sclerosis, and certain other disorders which are more appropriately considered in other sections.

1. ACUTE MYELITIS

Etiology and Pathological Anatomy.—As seen in childhood, the process is nearly always a diffuse one affecting especially the grey matter, and is identical in cause, lesions and symptoms with the poliomyelitis already described (Vol. I, p. 517). Exceptionally other causes may be active, such as an intoxication or an acute infectious disease, extension from a meningitis, or a trauma. The influence of syphilis is uncertain. The whole of the transverse section of the cord may be affected throughout a considerable extent of its length (*diffuse myelitis*), and the process even involve the brain as well (*encephalomyelitis*), although less often; or the inflammation may be in scattered foci (*disseminated myelitis*), or may affect the transverse section of the cord in a limited region only, and rarely completely (*transverse myelitis*). The lesions consist in redness of the cord from injection of the vessels and effusion of blood, and in softening of the substance. Later the red color is replaced by a yellow or whitish tint. Microscopically there are swollen axis cylinders; disorganization of the fibres; swelling and granular degeneration in the large nerve cells; leucocytic infiltration; granular material and masses of myelin and corpora amylacea; engorgement of the blood-vessels and thickening of their walls; and in older cases a sclerosing process, with increase of connective-tissue fibres.

Symptoms.—These vary greatly, depending upon the locality, the amount of the cord involved, or on whether the initial lesion remains localized or undergoes a rapid extension. As seen in transverse myelitis of the dorsal region, which may be taken as the type, there may early be moderate pain in the limbs or back, numbness in the limbs, a girdle sensation, and muscular twitching. Paralysis of the lower limbs is the most prominent symptom. It develops rapidly, and may be very decided in a few hours or days. It is of paraplegic type, and is combined with increase of reflexes, and later spastic symptoms; anesthesia; paralysis of the sphincters, and often the production of bed-sores. High fever, loss of appetite, debility, headache, and similar constitutional disturbances are usually present.

The occurrence of spastic symptoms, anesthesia, atrophy and altered electrical reactions depends upon the situation of the lesion in the cord. The tendon-reflexes are increased and there is rigidity and anesthesia, but no excessive atrophy or degenerative reaction, in the portions of the body receiving their nerve-supply from the cord *below* the seat of the lesion. On the other hand, atrophy is marked, degenerative reaction present, tendon-reflexes absent, and the paralysis is of the flaccid type in the parts supplied by the diseased portion of the cord, provided that the anterior horns are involved. Thus, when the lumbar cord is affected, the paralyzed legs are flaccid, anesthetic and atrophic, and exhibit reaction of degeneration but no increase of the knee-jerks. On the other hand, when the dorsal cord is diseased, the knee-jerks are exaggerated, the paralysis is of the spastic type, there is anesthesia, atrophy of the lower extremities is little marked, and the reaction of degeneration is absent. When the lesion is situated in the cervical region the arms exhibit paralysis and anesthesia; the paralysis finally becoming spastic and with increased reflexes if the lesion is above the origin of the brachial nerves, but flaccid and with atrophy and reaction of degeneration if the grey matter of the cord from which these nerves arise is involved.

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[1] Bruns, Cramer, and Ziehen, Handbuch der Nervenkrankheiten im Kindersalter, 1912, 456.

Course and Prognosis.—The course may be rapid ending in death from exhaustion or from complications, especially secondary nephritis and pneumonia. In other cases the symptoms may remain stationary, or show slight increase in their degree; and then improvement may begin after a few weeks. Final recovery may be complete, but is oftener only partial, especially if the disease has lasted several months without improvement of the paralysis.

Diagnosis.—The diagnosis is to be made from compression-myelitis by the more rapid onset and the absence of the earlier root-symptoms of that disease. In spinal hemorrhage the onset is sudden rather than rapid. In Landry's paralysis the sensation is not affected, and the paralysis indicates an ascending involvement of the spinal cord. Meningitis has more severe pain, hyperesthesia, and earlier rigidity.

Treatment.—The only treatment possible is absolute rest, with the patient preferably on the face; and dry cupping to the spine or the application of an ice-bag, if the case is seen early. The general strength and nutrition are to be maintained; and the greatest care used in the keeping of the skin of the back clean and the guarding against retention of urine. When improvement begins gentle massage may be employed.

2. ABSCESS OF THE SPINAL CORD

(Purulent Myelitis)

This is a very rare form of inflammation in early life. It may be due to trauma or oftener be the result of septic metastasis, or follow a purulent meningitis. Reference is not made here to the very numerous multiple collections of pus sometimes found in cerebrospinal fever and in poliomyelitis. The abscess is situated oftenest in the grey matter in the upper portion of the cord. The symptoms are vague, and usually overshadowed by those of the purulent meningitis or cerebral abscess which is liable to complicate the disease. In general they are those of a transverse myelitis. High, irregular fever and the presence of suppuration elsewhere in the body suggest the diagnosis. The course is rapidly fatal.

3. COMPRESSION MYELITIS

Etiology.—Any condition which produces decided compression of a portion of the spinal cord may bring about an inflammation at this point. The disease is, as a rule, a subacute or chronic process. Although it is sometimes seen as a result of pressure by tumor, trauma, or pachymeningitis produced in any way, much the most frequent cause is caries of the spine. It would be incorrect, however, to regard Pott's disease as synonymous with compression myelitis; for in the less severe cases of the former affection the process does not reach to the extent of actual involvement of the cord. The following description applies especially to the compression myelitis attending spinal caries.

Pathological Anatomy.—As a result of the disease of the vertebræ (see Spinal Caries, p. 442) a pachymeningitis is set up (see Pachymeningitis, p. 375), often with the production of an accumulation of pus; and this, together with, on the one hand, the narrowing of the spinal canal by curvature resulting from the destruction of the vertebræ and, on the other, the development of granulation tissue within the canal, produces a gradually increasing pressure upon the cord; generally exercised from in front, sometimes from one side. In the earlier stages there is only interference with the circulation, with consequent congestion and edematous infiltration. Later in old, severe cases there is an increase of interstitial tissue accompanied by degeneration of the nervous tissue, with more or less disappearance of the ganglion-cells and the nerve-fibres. The condition is such that the grey and the white matter can hardly be distinguished from each other. As a result of the compression the spinal cord is much flattened and is smaller than normal at the position of this. The extent of the lesion of the cord depends upon the number of vertebræ diseased. In most instances it does not affect more than 2 inches, and often less than this, although some minor degree of inflammation reaches both above and below the position of greatest involvement. In addition there is systemic degeneration, both ascending and descending, in certain tracts; a descending degeneration in the motor, an ascending one in the sensory. The nerve-roots coming from the region of compression are earlier and more extensively diseased than the cord itself, with corresponding degenerative changes in the nerves and the muscles supplied by them.

Symptoms.—These vary with the stage of the disease and with the position of the lesion. Only exceptionally is the process acute; as in cases due to trauma or to a sudden giving-way of carious vertebræ with consequent sudden pressure upon the cord. Among the earliest symptoms is pain, developing in the course of the nerves the roots of which are involved in the compression. This may occur spontaneously, or be elicited by pressure upon the region of the diseased vertebræ, or through motion of the spine. It may be constant or intermittent. Hyperesthesia is frequent, and later often localized anesthesia; together with muscular weakness, atrophy, and the electrical reactions of degeneration, all of these occurring in the same region as the pain. These symptoms, known as "root-symptoms," are, however, dependent upon the complicating neuritis, not upon the lesion of the cord itself.

The symptoms of the actual conpression-myelitis; *i.e.* the "cord-symptoms," are seen chiefly in regions of the body the nervous supply of which arises from the cord *below* the lesion. They are observed generally after the appearance of the root-symptoms. They consist first of all in paralysis coming on rapidly or much oftener slowly, the length of time depending upon the rapidity with which the myelitis develops. The paralysis is bilateral and of the spastic type, with increase of deep and superficial reflexes; pain; paresthesia; and often some degree of anesthesia, which in severe cases may be complete. There is no reaction of degeneration. There may be loss of sphincter-control.

Course and Prognosis.—The course of the disease varies greatly, and the prognosis likewise. When the principal lesion has been edema, there may be entire recovery after a few weeks under appropriate treatment. Even when there has been more or less destruction of the nervous tissue, regeneration may take place and the cord resume its power of conduction. In older cases, on the other hand, when the paralysis has been long-continued and when the sclerosing process has become well established, there is less hope of improvement. It is surprising, however, to what degree recovery may follow even after the disease has persisted for several years. (See Caries of the Spine, p. 444.)

Diagnosis.—This is usually not difficult, and depends upon the combination of the cord-symptoms and the root-symptoms as described. There is namely the earlier evidence of sensory disturbance, followed later by the paraplegia. Acute transverse myelitis exhibits a rapid development of paralysis. Tumors of the cord are of infrequent occurrence, and are less liable to produce pain on movement of the body than is caries of the vertebræ. The development of a characteristic curvature is conclusive proof of the existence of caries. In the absence of this condition the use of the *x*-ray may be of diagnostic aid.

Treatment.—This is chiefly that of the cause; operation being indicated for tumor and for trauma; while for spinal caries the treatment later to be described for this is to be employed.

LANDRY'S PARALYSIS

(Acute Ascending Paralysis)

It now seems certain that the majority of cases occurring in early life and called by this name were in reality but one of the forms of poliomyelitis (p. 529). Others probably were neuritis; others forms of myelitis. There are, however, still other cases rarely encountered which, for the most part, correspond clinically with the account first given by Landry,[1] and which warrant a separate description. The disease appears to be an acute toxic or infectious process, with a varying anatomical condition. In some instances no lesions whatever have been found; in others the early evidence of myelitis; in others involvement of the nerve-roots; and in still others microörganisms of different sorts. The enlargement of the spleen usually observed indicates the toxic-infectious nature of the malady.

Symptoms.—The symptoms consist often of an initial stage with malaise and fever followed by a progressive, ascending paralysis. This begins in the legs, is of the flaccid type, without electrical changes or trophic symptoms, and sensory disturbances are absent or little marked. The paralysis extends rapidly, attacking the trunk, upper extremities, and the respiration, speech, and swallowing, as the medulla becomes involved. The action of the sphincters is usually normal. Moderate fever may continue. Occasionally the upper extremities are first attacked, or the bulbar symptoms may even appear first and the advance be a descending one. The disorder reaches its full development in from 1 to 2 days, or sometimes longer. Death takes place in 1 or 2 weeks or a shorter time, often from asphyxia; yet cases of recovery are reported. The **diagnosis** is not difficult except that no clinical distinction exists between this Landry's paralysis and the ascending form of poliomyelitis. An ascending multiple neuritis is recognized by the presence of decided pain and tenderness in the nerve-trunks, the atrophy, altered electrical reactions, longer course, and favorable termination. The determination of the nature of the lesion is often impossible without autopsy.

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[1] Gaz. hebdom., 1859, VI, 472.

Treatment can be symptomatic only, and consists in absolute rest and the employment of supporting measures.

THE ATAXIAS

Ataxia [may exist as a symptom of various conditions, in some of which, as after infectious diseases, or in chorea, infantile cerebral paralysis, certain cases of tumor of the cerebellum or mid-brain, multiple neuritis and hydrocephalus, it is but one of other manifestations and perhaps of minor importance. In other diseases it constitutes the most striking symptom, and it is to these affections that some special attention must now be given. The most important of them is *Friedreich's ataxia.* Intermediate forms doubtless exist between this and certain other ataxic disorders, such as the *hereditary cerebellar ataxia* to be described. In addition to this latter, mention should be made in passing of the forms of *cerebellar ataxia* as classified by Batten.[1] The first of these is congenital cerebellar ataxia, depending upon imperfect development of the cerebellum or upon lesions analogous to those producing infantile cerebral paralysis. The second is acute cerebellar ataxia, which has already been described (p. 346) under the topic of acute encephalitis. The third is chronic progressive cerebellar ataxia, which is certainly closely allied to, or perhaps a form of, hereditary cerebellar ataxia.

1. FRIEDREICH'S ATAXIA

This disorder was first described by Friedreich in 1861.[2] Since that time a large number of studies have been made. In 1889 I published an analysis of 143 collected cases including 3 of my own;[3] but later studies would doubtless show that some of these belonged to the category of other diseases which exhibit ataxia. Many other instances have been reported since the date of this publication.

Etiology.—The direct cause is unknown. Hereditary or familial influence is a striking feature, a number of brothers or sisters being attacked, and sometimes the disease being directly inherited through several generations. In the family history reported by Carre[4] the grandmother; 9 of her children; and 7 of the children of one affected daughter suffered from the disorder. In other instances nervous diseases of some other sort have been present in the ancestors or have been encountered in other children of the family. The malady makes its appearance in early childhood, sometimes after puberty, and occasionally in infancy. The influence of acute, and usually infectious, disorders in precipitating the onset of the symptoms of Friedreich's ataxia is very decided.

Pathological Anatomy.—The process is a degenerative one, due, perhaps, to a congenital arrest of development, as a result of which sclerosing processes readily occur. It attacks especially the posterior columns of the cord, the lateral tracts, the direct cerebellar tracts, and the posterior nerve roots. Changes to a less extent may be found in other parts of the cord, especially in the columns of Clarke, and the lesions may extend in some degree to the medulla, but rarely above it. The cord is much diminished in size, and occasionally the medulla also. The cranial nerves are not often affected.

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[1] Brain, 1905, XXVIII, 484.

[2] Virchow's Archiv, 1863, XXVI, 391, 443; XXVII, 1; 1876, LXVIII, 145; 1877, LXX, 140.

[3] Transac. College of Phys., Phila., 1888, X, 196.

[4] De l'ataxie locomotrice progressive, 1865, 65; 248.

Symptoms.—The first symptom seen is ataxia, beginning in the lower extremities, and shown by uncertainty or staggering in the gait; very different from the undue forward propulsion of the legs in walking as seen in tabes. When standing the patient may sway from side to side (static ataxia), and when the eyes are closed the swaying increases in some cases (Romberg's symptom), while in others it does not. The sphincters are unaffected. The knee-jerks are absent. Trembling or oscillating movements of the legs when in certain positions are common. The feet often exhibit a characteristic deformity, consisting of high arching of the instep, shortening of the foot, and hyperextension of the great toe, sometimes with flexion of the last phalanx. This may be an early or a late symptom. Very gradually, sometimes only after several years, the process extends to the trunk and the upper extremities, and finally to the head. The patient now sways while sitting, and scoliosis is common. The hands exhibit marked ataxic movements on voluntary effort, the attempt at grasping being often peculiarly claw-like and over-reaching; and even while lying passive in the lap there may be choreiform or athetoid movements of the fingers. There is an irregular nodding or jerking movement of the head. The face develops a peculiar expressionless appearance. Nystagmus is common; optic atrophy rare; the pupillary reflexes are generally unaffected. The speech is jerking and peculiar, with alternation of pauses and of rapid enunciation of several words together. When the symptoms are well developed, the difficulty in walking increases to an entire disability, and in advanced cases there may be actual paralysis, both in the lower and upper extremities. Paralysis is, however, not one of the characteristic manifestations of the disease. There is no loss of control of the bladder and rectum.

Sensory symptoms are of little prominence in Friedreich's ataxia. Pain is uncommon, and probably accidental when present. The cutaneous sensibility is generally unaffected, sometimes slightly diminished, and the muscle-sense unimpaired. There are no trophic symptoms of moment, and muscular atrophy is usually not great even in well-developed cases. Vertigo is not infrequent. Salivation, disturbance of respiration, palpitation, and uncontrollable laughing are among rare symptoms. The intelligence is normal, although the vacuous expression and the difficulty in enunciation suggest the opposite of this. In comparatively few instances is there any real mental deficiency, and then far on in the course and occurring as a complication.

Course and Prognosis.—The course is slowly onward, although often with stationary periods. Occasionally the upper and lower limbs are affected almost simultaneously, and sometimes the speech as well. Generally, however, the upper extremities do not show involvement until from 1 to 5 years after the lower, and the affection of speech about 1½ years later still. To all this there are, however, many exceptions. In one of my cases 17 years elapsed between the involvement of the lower and of the upper limbs. The patient often reaches at last a state of comparative helplessness, and death may occur from asthenia or from some intercurrent disorder. Life may, however, be prolonged for many years.

Diagnosis.—From *tabes dorsalis* the disease is distinguished by the characteristic uncertain gait, nystagmus, the early age of onset, and the family history; and by the absence of severe pain and of other sensory manifestations, pupillary symptoms, optic atrophy, and visceral disturbances. Friedreich's ataxia is frequently supposed to be *chorea*, and I have more than once seen instances of this error. Chorea, however, is of more sudden onset, generally involves the arms first and most decidedly, and is lacking the club-foot, scoliosis, and nystagmus of Friedreich's ataxia. It is, moreover, an acute disease. *Disseminated sclerosis* usually develops later in life; the speech is scanning rather than irregular and jerking; the knee-jerks are increased; there is intention-tremor; the nystagmus is more extreme; spastic paralysis may develop, and the intellect is oftener affected. *Cerebellar tumor* gives a more typically drunken gait, and there is headache, decided vertigo, and optic atrophy; while the tendon-reflexes are not abolished. The distinction from *hereditary cerebellar ataxia* will be considered when discussing that subject.

Treatment.—This is entirely unavailing and can be only symptomatic. The general nutrition must be sustained; and when scoliosis and the inability to sit become marked the application of a plaster or other jacket to the thorax may give relief.

2. HEREDITARY CEREBELLAR ATAXIA

Etiology and Pathological Anatomy.—Until the publication by Marie[1] in 1893, cases of this nature were generally classified under Friedreich's ataxia; and studies by many investigators show that the clinical distinctions cannot always be sharply drawn, and that there are undoubtedly intermediate forms. From a pathological standpoint, too, there is no uniformity in the confinement of the lesion to the cerebellum, but the cord may also be involved. It would appear that the two diseases may properly be regarded as but different types of the same disorder, which might well be denominated Hereditary Ataxia. In the one the spinal symptoms predominate (Friedreich's ataxia); in the other the cerebellar (Cerebellar ataxia). The following description applies to the latter, the hereditary cerebellar ataxia of Marie.

A very prominent etiological factor is familial tendency, shown as in Friedreich's ataxia. In the large majority of instances the disease begins after the period of childhood; yet cases commencing in early life have been reported. The anatomical lesions are seen chiefly in the cerebellum, which is unusually small and exhibits degeneration of the cortex and the fibres connecting it with the central nuclei; while the spinal cord is not involved. To this, there are, as stated, exceptions, and there are reported cases in which the cord was small and the cerebellar tracts and other regions were degenerated; while in still others the cerebrum has been found hypoplastic.

Symptoms.—These consist in a gradually increasing ataxia of a peculiar nature. The gait is more of the drunken cerebellar type than in Friedreich's ataxia, although this is open to exceptions, and walking finally becomes impossible. The arms likewise partake of the ataxic condition, and choreiform movements here are marked, combined also with irregular oscillation of the head and trunk. The tendon-reflexes are increased, and ankle-clonus is frequent. The scoliosis and the deformity of the feet, often seen in Friedreich's ataxia, seldom develop. Nystagmus may occur; oculomotor involvement, such as ptosis, diplopia, and strabismus, is not uncommon; and optic atrophy is a frequent symptom. The pupillary reflex may be involved. Speech becomes irregular, slow and indistinct. The sphincters are not affected, and the intellect is at first normal but is liable to be disordered as the disease develops. Derangements of sensation are usually absent, but are encountered oftener than in Friedreich's ataxia. The course is slow and the prognosis unfavorable and uninfluenced by treatment.

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[1] La sem. méd., 1893, XIII. 444.

Diagnosis.—The diagnosis from Friedreich's ataxia is impossible in the mixed forms. In typical cases it rests especially upon the increase of the knee-jerks, ankle-clonus, cerebellar gait, oculo-motor paralysis, and optic atrophy. Disseminated sclerosis likewise exhibits increased knee-jerks; but shows, too, very decided intention-tremor, typically scanning speech, periods of temporary improvement, and symptoms pointing to various different foci of disease. Treatment is unavailing.

3. TABES DORSALIS

(Locomotor Ataxia)

Etiology and Pathology.—Tabes dorsalis, or locomotor ataxia, admitted to be the result of syphilis, is a disease limited so largely to adult life and with symptoms so characteristic that very brief mention need be made of it here. Rarely it does appear at an early period. Marburg in 1903[1] collected 34 such cases, and Cantonet[2] in 1907, 88, and the number has been increased since then. The great majority of these have developed, it is true, only after puberty (*Juvenile tabes*); yet cases have been seen with symptoms of tabes appearing as early as the age of 5 years. Females are as frequently affected as males. Oftenest the syphilis upon which the disease depends is hereditary in origin, but sometimes it may have been acquired early in life.

Studies upon the pathological anatomy have been limited to very few cases, and all of these have shown complicating lesions in addition to those characteristic of the disease itself.

Symptoms.—The symptoms are similar to those seen in adult life with, however, certain differences. The onset, namely, is more indefinite and deceptive; the characteristic ataxic gait is less marked; the sensory manifestations, such as lancinating pain, paresthesia, anesthesia, and the like, are less prominent; and paralysis of the ocular movements somewhat less often seen. On the other hand, optic atrophy is an unusually frequent and often the earliest symptom. Disturbances in urination are among the early manifestations. A combination of the evidences of tabes with those of general paralysis is not uncommon. The course and prognosis are as in adult life.

Diagnosis.—This is usually easy, based upon the symptoms and the syphilitic history. Friedreich's ataxia is distinguished in many ways, among them being the familial history often present, nystagmus, disturbance of speech, prominence of ataxic symptoms, and the absence of optic atrophy. Hereditary cerebellar ataxia has practically no resemblance. The ataxia of this disease is much more marked, and the knee-jerks are normal or increased. The treatment is similar to that for the affection in adults, and is purely symptomatic.

HEREDITARY SPASTIC PARAPLEGIA

Spastic paraplegia arises from numerous causes, such as cerebral diseases, caries of the spine, tumors, spinal pachymeningitis or lepto-

[1] Wien. klin. Woch., 1903, XVI, 1295.

[2] Arch. d'ophthalm., 1907, XXVII, 708.

meningitis, etc. There exists, however, a distinct form of which brief notice must be taken, in which the familial tendency is very marked; as pointed out by Strümpell,[1] Newmark,[2] Erb[3] and others. Bayley[4] reports a family in which the disease appeared in 5 generations, developing in all those affected at about the age of 5 years; and Spiller[5] an instance of 14 cases in one family. The disease may begin at any age, although oftener in early life, and in either sex. The lesions consist in degeneration of the crossed pyramidal tracts; sometimes to a slight extent of the direct cerebellar tracts, and of the columns of Goll.

Symptoms.—The symptoms come on gradually, the first being difficulty in walking owing to the development of a spastic condition which causes the feet to be pushed stiffly along the floor. The knee-jerks are increased, ankle-clonus is present, and the muscles are rigid. There is no loss of sensation, and the sphincters are not involved. A final inability to walk is not the rule, and there is actual loss of muscular power only in the last stages if at all. The arms are only exceptionally affected. Bulbar symptoms are not seen in typical cases, and their presence indicates some complicating condition. The intellect is normal.

The **course** is very prolonged, for although the symptoms may reach their height with comparative rapidity, they may then long remain unchanged. The prognosis is unfavorable for recovery, although the duration of life is not curtailed. The **diagnosis** is to be made only from other conditions which may produce paraplegia; and this cannot be done with certainty unless there is evidence of a familial occurrence. The existence of any defect of intelligence points to a spastic paraplegia of cerebral origin, and the presence of bulbar symptoms suggests a disseminated sclerosis. The disease is little influenced by treatment, although systematic exercises of a suitable nature have occasionally been followed by improvement.

THE MUSCULAR ATROPHIES AND DYSTROPHIES

Although some of these appear to be entirely or primarily purely muscular diseases, and would be described more properly under Diseases of the Muscles, others are certainly spinal in origin and others perhaps neural; and the inter-relationship of all seems so close in their clinical manifestations, and, to a certain extent, in their pathological, that they may well be studied together. Those conditions are omitted in which the atrophy is but a minor symptom of some more general nervous disorder.

The different types are conveniently divided into: (*A*) *Spinal muscular atrophy* or *amyotrophy*, which may be subdivided into (1) Amyotrophic lateral sclerosis; (2) Progressive muscular atrophy of the hand-type; and (3) the Werdnig-Hoffmann type. (*B*) *Neural muscular atrophy.* (*C*) The *primary myopathies*, or *muscular dystrophies*, of a number of types presently to be considered. Yet intermediate forms between the groups, and especially between members of one group, undoubtedly occur, and no classification can be absolute. Many of the varieties have certain features in common; chiefly among them being, in the first place, a distinct familial tendency, and, in the second, a slowly progressive course. Some of them are peculiarly characteristic of early life; others are seen principally or only in adult life.

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[1] Archiv f. Psych., 1880, X, 711. Deutsche Zeitschr. f. Nervenheilk., 1893, III, 495.

[2] Amer. Journ. Med. Sci., 1893, CV, 432.

[3] Deutsche Zeitschr. f. Nervenheilk., 1895, VI, 137.

[4] Journ. Nerv. and Ment. Dis., 1897, XXIV, 697.

[5] Phila. Med. Journ., 1902, IX, 1129.

A. PROGRESSIVE SPINAL MUSCULAR ATROPHY

1. Amyotrophic Lateral Sclerosis (*Charcot's Type of Muscular Atrophy*).[1]—This is a disease chiefly confined to adults, although a few cases beginning in childhood have been reported. In some of these there has been shown a very decided familial tendency, several sisters or brothers having been attacked, as in the instances reported by Seeligmüller,[2] in which 4 children of one family showed symptoms in infancy. Beyond this the cause is unknown. The pathological **lesion** is a degeneration of the pyramidal tracts and of the anterior horns of the spinal cord. The **symptoms** in brief consist in an atrophic paralysis, yet with a spastic condition; the atrophy being seen especially in the arms, and the spastic manifestations in the legs. The loss of power and the spastic symptoms appear before any atrophy shows itself, this being in sharp contrast with the relations obtaining in the hand-type of progressive muscular atrophy. Contractures develop, with pain on movement, increase of the tendon-reflexes, and fibrillary twitchings. The patient at last becomes helpless. Bulbar symptoms finally appear, with interference with speech and swallowing. The **course** of the disease is slow, lasting over several years, and the **prognosis** is unfavorable and uninfluenced by treatment.

2. Progressive Muscular Atrophy; Hand-type (*Duchenne-Aran Type*).—This malady was brought into prominence through the writings especially of Duchenne[3] and of Aran,[4] from whom the variety often takes its name. The disease is not common at any time of life, and is of rare occurrence before puberty. In 210 cases collected by Seidel[5] 44 began before the age of 15 years, and 28 of these between 1 and 10 years. Males are much more frequently attacked than females. A familial tendency is sometimes seen, but is oftener not in evidence.

The **lesions** consist in degeneration of the anterior horns of the spinal cord with secondary involvement of the nerve-roots and of the muscles.

Symptoms.—The symptoms are characteristic. Atrophy begins in the muscles of the hands; first in the thenar eminences and then the hypothenar and the interossei, and extends thence to the forearms. Here it may cease; or may spread to the arms, shoulders, neck, back, and finally, late in the disease, to the lower extremities. The claw-like appearance of the hand is very characteristic (*main en griffe*). Exceptionally the disease attacks other localities before the hands and arms. The fibrillary twitchings which the muscles exhibit, sometimes before atrophy is seen, constitute a very striking symptom. The faradic contractility disappears gradually in proportion to the amount of atrophy present; and in well-advanced cases there may be a reaction of degeneration. The tendon-reflexes are diminished or absent.

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[1] Charcot and Joffroy, Arch. de physiol. norm. et path., 1869, II, 334.

[2] Deut. med. Wochenschr., 1876, II, 185.

[3] Atrophie musculaire avec transformation graisseuse 1849. Ref., in his l'électrisation localisée, 1855, 814.

[4] Arch. gén. de méd., 1850, XXIV, 5.

[5] Gerhardt's Handb. der Kinderkrank., 1889, V, 2, 9; 10.

The **course** is very slow but progressive, the wasting being only gradual, and perhaps some years passing before the lower extremities become involved. Finally the patient grows powerless and wasted to an excessive degree.

Diagnosis.—The disease is to be distinguished from amyotrophic lateral sclerosis by the absence of spastic symptoms, and, still more important, by the fact that the atrophy precedes the paralysis, the latter being in proportion to the degree of the former. It is to be observed, however, that the differences between the two diseases are not always sharply defined, and that some writers class them together.

Treatment is without avail, but the malady is fatal only through complications; except that in certain instances the paralysis involves the tongue and the muscles of respiration, making swallowing and coughing difficult, and bringing about a fatal ending.

3. The Werdnig-Hoffmann Infantile Type of Progressive Muscular Atrophy.—This very unusual disorder described by Werdnig,[1] Hoffmann[2] and others, exhibits lesions similar to those seen in the Duchenne-Aran type just described, but has certain very definite distinguishing clinical features. Parsons and Stanley[3] found but 21 well-authenticated cases in medical literature, and Ziehen,[4] places the number at over 30. Foremost among the characteristics is the striking familial history, nearly all of the cases having occurred several in one family of brothers and sisters, and sometimes through a number of generations (Hoffmann). It begins usually in the first, sometimes the second, year.

Symptoms.—The symptoms develop first in the lower extremities or the trunk, and consist in progressive weakness and atrophy; and this spreads to the neck, shoulders and last to the arms, hands, and toes, the order being almost the reverse of that seen in the Duchenne-Aran type. The tendon reflexes are abolished; the limbs are flaccid; fibrillary twitchings are sometimes seen. The cranial nerves are not affected or but slightly so; the sensation is not disturbed; the sphincters are not involved; the electrical contractility is much diminished or abolished, and the reaction of degeneration may sometimes be present. The extension and increase of the paralysis is rather rapid, and the patient soon becomes helpless, greatly wasted, and variously deformed; and death takes place usually in from 1 to 5 years through involvement of the respiratory muscles and consequent disease of the lungs, or through some other complication. In exceptional cases life has continued for a longer period.

Diagnosis.—This is to be made from the Duchenne-Aran type especially by the familial occurrence, early age and the sequence of symptoms. It is certain, however, that the two types are very closely allied, and might with propriety be classed as the same affection with varying manifestations. From some forms of hereditary muscular dystrophy unattended by hypertrophy the diagnosis is difficult, but the course in the Werdnig-Hoffmann type is much more rapid and the onset earlier. Amyotonia congenita is distinguished especially by the still earlier age of onset, the much less degree of atrophy, the slower course, and the tendency to improvement sometimes seen.

[1] Archiv f. Psych., 1890, XXII, 437.

[2] Deutsche Zeitschr. f. Nervenheilk., 1893, III, 427.

[3] Brain 1912, XXXV, 50.

[4] Bruns, Cramer and Ziehen; Handb. der Nervenkr. im Kindersalters, 1912, 415.

B. (4) PROGRESSIVE NEURAL MUSCULAR ATROPHY

[Peroneal Type

To this form the title of "Type of Charcot-Marie-Tooth" is often applied.[1]

Etiology.—It is an infrequent variety, of a very positive familial and hereditary nature, occurring sometimes through several generations. It was seen, for instance, in 20 individuals through 4 generations as recorded by Herringham.[2] It is rather more common in males, and develops oftenest in early childhood, but may first appear in later childhood or even not until after puberty. The disease seems to occupy a midway position between the spinal muscular atrophies and the muscular dystrophies.

Pathological Anatomy.—The lesions are not certainly established, since conditions have been described varying with the case. The most frequent are degeneration of certain of the nerves, with interstitial changes; simple atrophy of the muscles; and sclerosis of the posterior columns and to a slight extent of the lateral tracts and of the anterior horns. Not all of these changes have been present in every case examined; and which of them may be primary is not yet determined. The title "neural" is therefore not strictly speaking applicable as describing any necessary characteristic.

Symptoms.—The first symptoms in typical cases consist in wasting and loss of power in the peroneal muscle-group and the extensors of the toes of both legs; with consequent walking upon the outer side of the foot, foot-drop, high-stepping gait, and later club-foot in the equinus or equino-varus position. As the disease progresses the atrophy spreads to the rest of the muscles below the knee, and the ankle-jerks are diminished; but the knee-jerks are unaffected unless the muscles of the thigh become involved. Fibrillary twitchings are common. The electrical response is diminished, and there may be a partial reaction of degeneration. In some cases there are such sensory disturbances as pain, hyperesthesia or anesthesia.

Sometimes antedating or simultaneously with the involvement of the legs, but oftener only after from 1 to 4 years later, the disease may appear in the hands, with the production of the claw-hand, and perhaps in the forearms also. The power of walking is retained, since the muscles of the thigh are involved only late if at all. The trunk, shoulders, upper arms, neck, and face usually escape entirely. The course of the disease is very slow and often with long intermissions; and the prognosis for recovery is unfavorable, although little change may take place for years.

Diagnosis.—The diagnosis is to be made from progressive muscular atrophy of the hand-type by the commencement in and limitation early to the lower extremities, and the marked familial tendency. The Werdnig-Hoffman type begins earlier, attacks the legs and pelvis first instead of the feet, and runs a more rapid course. The muscular dystrophies oftenest commence in the muscles of the trunk or shoulder girdle, not in the muscles below the knee, and disturbances of sensation do not occur. Multiple neuritis produces much greater sensory disorder; has no familial history; if beginning in the feet soon shows itself elsewhere as well; is of much more rapid development and course, and offers a more favorable prognosis. **Treatment** is of no avail in restoring damage once done; but much may often be accomplished in preserving muscles as yet unaffected, and in maintaining the ability to walk by the employment of massage and electricity and of suitable orthopedic apparatus, tenotomy, and the like.

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[1] Charcot-Marie, Revue de méd., 1886, VI, 97. Tooth, The Peroneal Type of Progressive Muscular Atrophy, Grad. Thesis Cambridge, 1886.

[2] Brain, 1888, XI, 230.

C. THE MUSCULAR DYSTROPHIES

The remaining forms of muscular atrophy are to be classed together under the title "Muscular Dystrophies," or "Primary Myopathies." The different varieties of these have many points in common which distinguish them from the other types of atrophy already described. All begin before adult life, and the majority of the forms in childhood, and a familial or hereditary tendency is often observed. Atrophy is decided in all, although a pseudohypertrophy or actual hypertrophy is an early and prominent symptom in one variety. The disease is first manifested in other regions of the body than the distal portion of the extremities; fibrillary twitching, qualitative electrical changes and disturbances of sensation are absent; and in typical cases the nervous system does not exhibit lesions. Various types have been described not sharply differentiated from each other, and showing, often, intermediate forms. The principal ones to be mentioned here are (5) the pseudohypertrophic, (6) the simple atrophic, (7) the facio-scapulo-humeral, and (8) the juvenile type.

5. Pseudohypertrophic Type of Muscular Dystrophy (*Pseudohypertrophic Paralysis*). **Etiology.**—This is one of the more frequent and most easily recognized varieties. Although isolated cases may be met with, the hereditary and familial tendency is very marked, frequently several children in one family being effected; the males much oftener than the females, and the inheritance coming through the mother, herself often not a sufferer from the disease. The majority of cases begin in early childhood; seldom after puberty; and sometimes it has been noted that the children never have learned to walk quite normally.

Pathological Anatomy.—The lesions are limited to the changes in the muscles. They consist at first of hypertrophy of the individual muscle-fibres, at least in certain muscles; and, according to Erb,[1] in all the affected regions. This is followed by atrophy, with loss of transverse striation; splitting; vacuole-formation; increase of muscle-nuclei; and, finally, fatty degeneration. Hypertrophic and atrophic fibres are found in the same muscle, the number of the latter gradually increasing until the whole muscle is more or less completely degenerated. The increase of size in some of the muscles at first is due in part to actual hypertrophy, but later entirely to pseudohypertrophy depending upon increase of connective tissue and infiltration by fat, the degree of each of these varying with the group of muscles involved.

The description of the pathological lesions as just given applies to all the muscular dystrophies, with the exception that in those of types other than the one now under consideration the lipomatosis and connective-tissue hypertrophy is not so great. In these types no visible macroscopic pseudohypertrophy is produced, and the actual hypertrophy is of a transitory and inconspicuous nature. The muscular dystrophies exhibit no lesions in the spinal cord or the nerves, at least in the majority of instances; and although in some advanced cases changes in the anterior horns have been found, it is uncertain whether or not these are of a secondary nature.

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[1] Deutsch. Zeit. f. Nervenheilkunde, 1891, I, 13.

Symptoms.—The onset is insidious. Sometimes the gait has always been unsteady and uncertain; but generally there develops slowly an increasing weakness in the lower extremities in children previously normal, with a tendency to fall readily; and a difficulty in rising from the floor or from a chair or in climbing the stairs. The gait is waddling, and while standing there is lordosis, with the trunk very rigid or inclined backward. The peculiar and very characteristic manner of rising from the floor is shown in the accompanying illustrations (Fig. 354).

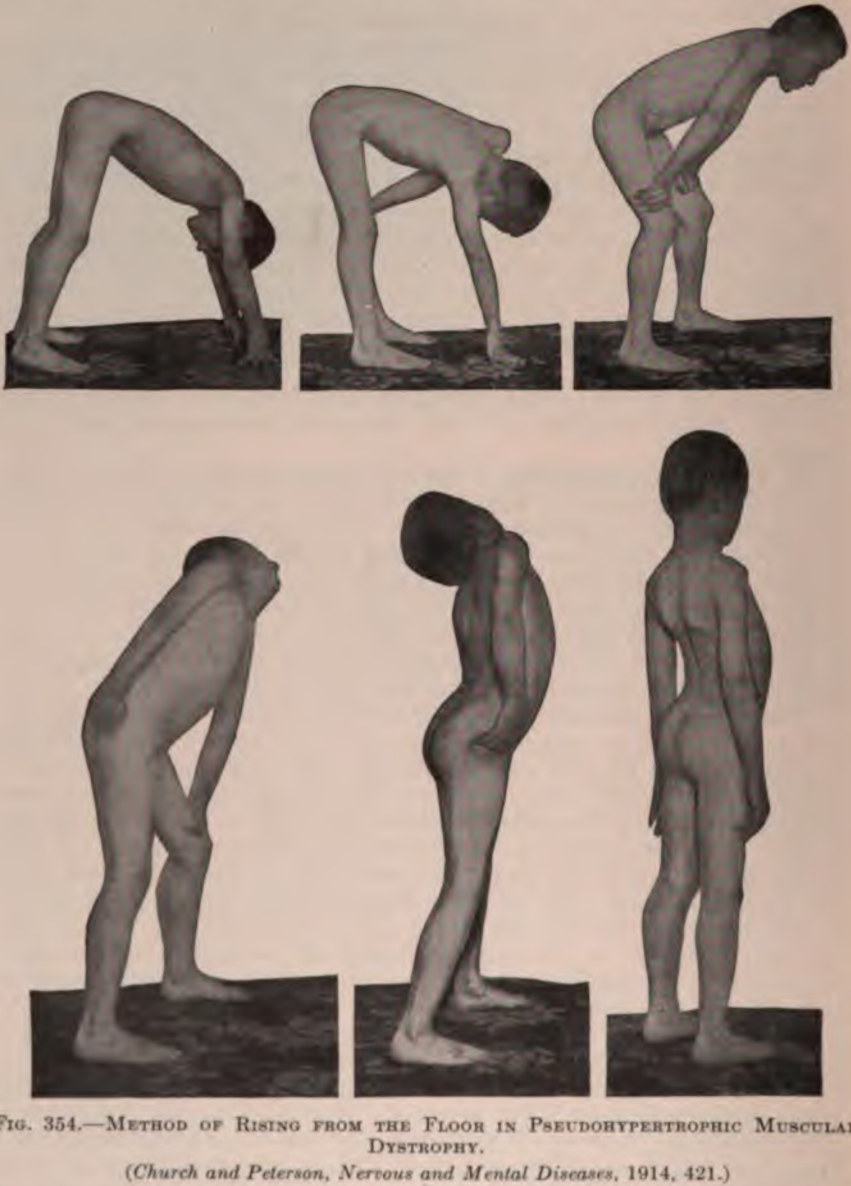

FIG. 354.—METHOD OF RISING FROM THE FLOOR IN PSEUDOHYPERTROPHIC MUSCULAR DYSTROPHY.

(*Church and Peterson, Nervous and Mental Diseases*, 1914, 421.)

With the weakness there is noticed a symmetrical increase in the size of certain of the muscles. This is usually especially well seen in the calves, which may attain a remarkable development (Fig. 355). The glutei, too, are often very large, and occasionally other muscle groups of the lower extremities may be affected. In the upper extremities an enlargement of the infraspinati is frequent, combined often with a similar change in the supraspinati, deltoids, and sometimes the triceps. In contra-distinction to these changes atrophy is commonly observed in other muscles, notably the flexors of the thigh, latissimi dorsi, serrati, biceps and the lower part of the major pectorals. A hypermobility of the shoulder develops on passive movement, shown especially by the manner in which the arms and shoulders slip upward and through the hands placed in the armpits in the attempt to lift the child. The distribution of atrophy and of hypertrophy detailed represents the general rule, but in some cases other muscle groups may be involved chiefly or in addition to these. Sometimes even the tongue may be hypertrophied, but, as a rule, the face is not involved. I had under my care a patient dying in adoles-

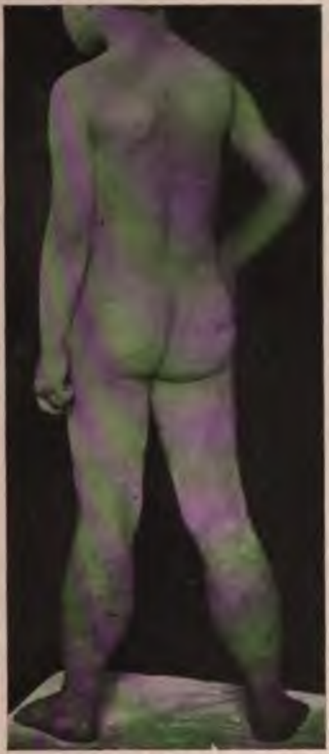

FIG. 355.

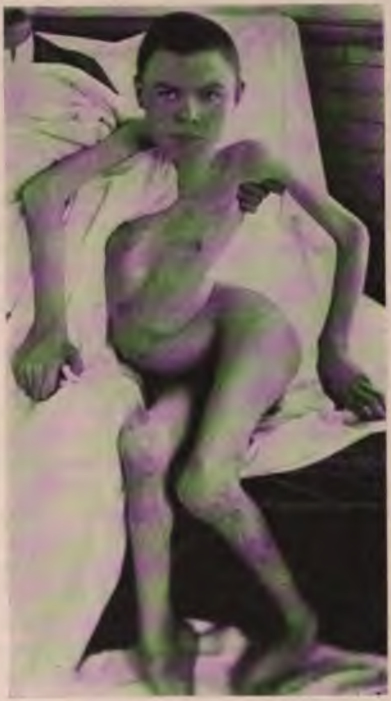

FIG. 356.

FIG. 355.—PSEUDOHYPERTROPHIC MUSCULAR DYSTROPHY.

Showing enlargement of the calves. (*Courtesy of Dr. Howard C. Carpenter.*)

FIG. 356.—PSEUDOHYPERTROPHIC MUSCULAR DYSTROPHY, ADVANCED CASE.

Boy of 15½ years, in whom the disease began at about the age of 7 years. (*Griffith, Univ. Med. Magaz.*, 1889, *Feb.*)

cence where extreme wasting was almost universal[1] (Fig. 356) except that the tongue was phenomenally large.

This combination of hypertrophy and atrophy, together with the lordosis which is usually present on standing, causes the calves, the buttocks, and the shoulder-blades to stand out with unusual prominence. The very large muscles, although looking powerful, are in reality weak. The tendon-reflexes are unchanged at first, but later grow less active and at last disappear. The superficial reflexes are undisturbed; fibrillary contractions are absent; the electrical response gradually diminishes, but there is no reaction of degeneration. There are no disturbances of sensation and the sphincters are unaffected. The intellect is normal, but to this there are not infrequent exceptions.

Course and Prognosis.—The course of the disease is very slow, often with periods of intermission. It is more rapid in cases developing early, and generally advances more slowly after puberty is passed. Gradually power is lost more and more, the patient becomes bed-ridden, and in long-continued cases the wasting of most of the muscles of the limbs becomes very complete; all signs of pseudohypertrophy disappear; the patient is able to do little more than move his hands; and various deformities result from contraction of the muscles. Death is likely to occur from some complicating affection before adult life is attained.

Diagnosis.—The diagnosis rests upon the features detailed as characteristic of the dystrophies in general; the peculiar gait; the method of rising from the floor; and the presence of pseudohypertrophy. In the far-advanced cases the diagnosis of this type of dystrophy is impossible except from the history, since all pseudohypertrophy may have disappeared. In the beginning of the disease, too, it is difficult or impossible; for although awkwardness shown in rising from the floor is suggestive, this can occur to some extent in other diseases, such as spinal caries as well as in other forms of muscular dystrophy.

6. The Simple Hereditary Atrophic Type (*Hereditary Muscular Atrophy, Leyden*[2]*-Möbius*[3]).—This less common form exhibits a remarkable development of the familial and hereditary tendency. It begins usually at from 8 to 10 years of age. The earliest symptoms are atrophy and weakness in the muscles of the back and lower extremities. There is lordosis and difficulty in walking. Evidences of pseudohypertrophy are never seen, but in other respects the symptoms are very similar to those present in the pseudohypertrophic form. The disease advances very slowly, involving finally the muscles of the upper extremities, as in the case of the pseudohypertrophic type. It is questionable, indeed, whether the two should not be classed together, cases of Leyden's type being merely those in which the pseudohypertrophy has been transitory and overlooked.

7. The Facio-scapulo-humeral Type (*Type of Landouzy and Déjérine*).—Although previously described by Duchenne,[4] this infrequent form of myopathy usually bears the names of Landouzy and Déjérine.[5] The familial and hereditary characteristics are usually very prominent. The disease begins oftenest in childhood; sometimes as early as 2 years of age, or possibly even before this, although escaping recognition. It may, however, not appear until after puberty or even in adult life.

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[1] Univ. Med. Mag., 1889, Feb.

[2] Rückensmark's Krankheiten, 1876, II, 2, 525.

[3] Volkmann's Sammlung, 1879, No. 171.

[4] L'électrisation localisée, 1855, 833. Arch. gén. de méd., 1868, XI, 421.

[5] Rev. de méd., 1885, V, 81.

Symptoms.—The changes are seen first in the facial muscles, and earliest here in the orbicularis oris and orbicularis palpebrarum. As the disease develops the face becomes peculiarly impassive and expressionless; the eyelids cannot be tightly closed, the eyebrows lifted, or the forehead wrinkled. The appearance does not change with laughing or crying. There may be difficulty in sucking, but not in chewing or swallowing; while older children cannot whistle and speech may be indistinct (Fig. 357). The muscles undergo marked atrophy, although the lips do not suffer from this but are prominent and thickened, the upper extending beyond the lower (*tapir-mouth*). The progress of the disease may stop here for a while, generally for from 5 to 15 years but sometimes for a short time only, before it advances to the muscles of the shoulder girdle and the upper arms. It spares for the most part the infraspinati, supraspinati, and the muscles of the forearm. Later those of the pelvis and lower extremities may become involved. The atrophy of the shoulder girdle causes the scapulæ to stand out in wing-form; but other deformities, as of the hands and feet, are seen only in very long-continued and advanced cases. Fibrillary contractions are absent. There is diminished electrical contractility but no reaction of degeneration. The course is one of the slowest seen in the muscular dystrophies, and as a result little, if any, difficulty in walking or in rising from the floor is usually observed. For the same reason the patellar reflexes are long intact. It may be 20, 30, or more years before any tendency to extension of the disease is shown. On the other hand, in some cases the spread is more rapid, and the patient may be almost helpless before adult life is attained.

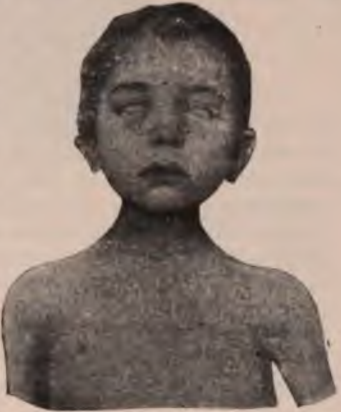

FIG. 357.—FACIO-SCAPULO HUMERAL TYPE OF MUSCULAR DYSTROPHY.

Showing the tapir-mouth and the difficulty in closing the eyelids. (*Haushalter, Revue de méd.*, 1898, *XVIII*, 447.)

8. The Juvenile Type of Muscular Atrophy (*Erb's Type; Scapulo-Humeral Type*).—Under this title are included cases as first described by Erb[1] in which the paralysis, usually symmetrical, begins in the shoulder girdle, leaving the face unaffected. It is probably the least frequent variety. Hereditary and familial relationships are present as in other forms. Although it may develop in childhood or even before this, its commencement is oftenest first seen at or soon after puberty or early in adult life.

Symptoms.—The scapulæ become markedly winged from the affection of the serrati magni and trapezii, and there is difficulty in raising the arms above the head. Other muscles involved are the pectorals, latissimi dorsi, rhomboids, and later the muscles of the upper arm. All there are not only paralyzed but atrophic; while the deltoids, infraspinati and supraspinati are not affected until very late if at all, and are even hypertrophic. The muscles of the forearms and hands escape. Disturbances of sensation, fibrillary contractions, and reaction of degeneration are absent. The tendon-reflexes are at first unaltered; later diminished in proportion to the degree of atrophy.

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[1] Ziemssen's Handb. der allgem. Therap., 1882, III, 389; Deut. Archiv für klin. Med., 1884, XXXIV, 467.

Sometimes the pelvis and thighs are attacked simultaneously with, or before, the upper portion of the body, the muscles affected being especially the quadriceps extensors, the adductors, and the iliac and psoas groups. As a result the patient is unable to flex the thigh easily upon the abdomen. As a rule, however, the upper part of the body is that first involved, and the lower not until much later.

The **course** of the disease is slow, often with periods of long intermission in the progress which perhaps lasts several years. The **diagnosis** rests upon the age of development and the muscular regions first involved. The disease is very closely allied to the facio-scapulo-humeral type, differing from it in the later age at which it appears and in the freedom from atrophy of the face.

AMYOTONIA CONGENITA

(Myatonia Congenita)

The exact relationships of this disease, first described by Oppenheim,[1] are not yet satisfactorily understood. The close affinity to the muscular dystrophies and possibly also to diseases of the spinal cord make it necessary to consider it in this connection. It was at first supposed to be a condition of atony rather than of atrophy, but later investigations show very distinct lesions in the muscles and sometimes in the spinal cord as well. Batten[2] groups it among the muscular dystrophies. In an investigation published in 1910[3] I collected 49 cases including 1 of my own; and later, with Spiller,[4] added 11 others, and the pathological affiliations were studied. Other cases have since been reported, Faber[5] raising the number of recorded cases to 115.

Etiology and Pathological Anatomy.—The cause is entirely unknown. Hereditary influence appears to be absent. The disease is a congenital one; at least this is true in nearly all reported instances. The lesions have shown considerable variation and it is very possible that more than one pathological condition accounts for the clinical picture of amyotonia congenita. The alteration in the muscles may be slight, or there may be very decided diminution of the number of muscle-fibres, combined with extensive lipomatosis and sclerosis. In some severe cases the spinal cord and the nerves are involved as well, the chief change in the former being a reduction in the number of cells in the anterior horns, and in the latter reduction of the size, or even atrophy, of the anterior nerve roots and of certain of the nerves. Yet in the matter of both the muscular and the nervous lesions it is not certain whether the changes are degenerative, or dependent upon a failure of development.

Symptoms.—These consist of remarkable flaccidity and loss of power, seen most of all in the lower extremities, but extending more or less over the whole body. The weakness may be so great that the limbs are almost powerless except for slight movement of the fingers and toes. A very few patients retain the ability to stand. In the severer cases the muscles of the trunk and of the neck and the intercostal muscles share in the condition; and as a result the child cannot sit or hold the head erect, and respiration may be interfered with to some extent. Any movements possible are made with peculiar slowness, atony and uncertainty, yet without any ataxia; and there is often seen a remarkable degree of passive hypermobility of the joints (Figs. 358, 359 and 360). Sensation is not disturbed; the electrical contractility is much diminished or absent, but without reaction of degeneration; the sphincters are not involved; the patellar reflexes are usually diminished or absent; the face is rarely attacked; the intelligence is normal; there are no fibrillary contractions; the muscles do not appear to be atrophied, although they are flaccid and soft; the general nutrition is well preserved, and contractures of some of the muscles are not infrequent.

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[1] Monatssch. f. Psychiat. u. Neurol., 1900, VIII, 232.
[2] Quarterly Journ. of Med., 1910, III, 313.
[3] Archiv f. Kinderh., 1910, LIV, 241.
[4] Amer. Jour. Med. Sci., 1911, Aug.
[5] Amer. Jour. Dis. Child., 1917, XIII, 305.

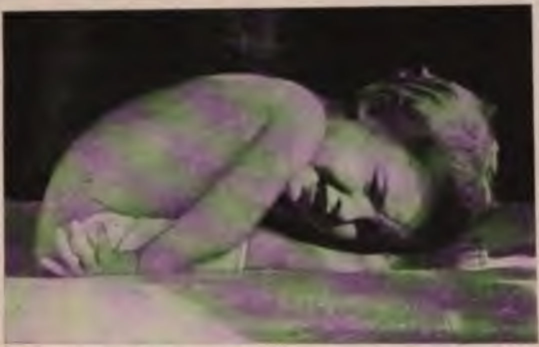

Fig. 358.—Amyotonia Congenita.

Boy of 15 months, in the Children's Ward of the Hospital of the University of Pennsylvania. Admitted Dec. 8, 1909. Never able to sit up, or hold head erect. Voluntary movement of the limbs very slight, moderate passive hypermobility. Improved decidedly under treatment, but developed pneumonia and died Oct., 1910.

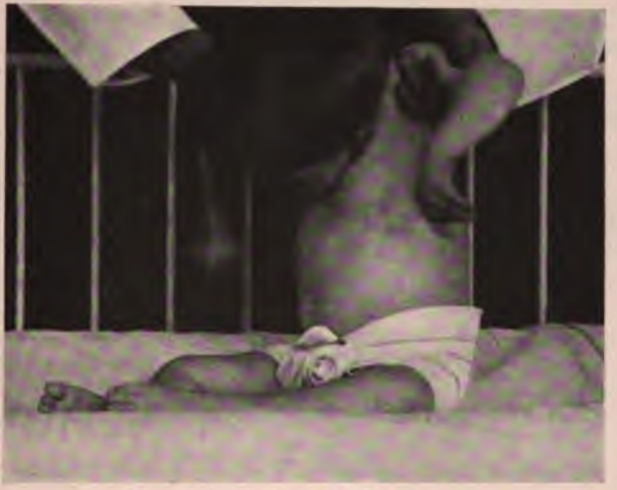

Fig. 359.—Amyotonia Congenita.

Same case as in Fig. 358. Shows hypermobility at the shoulder-joint.

Course and Prognosis.—These are not yet well determined. As far as observations indicate, there is no tendency for the condition itself to grow worse, and in a number of cases a decided improvement has been seen; but no instance of complete recovery has been as yet recorded. There certainly seems no tendency for the' loss of power to increase. The mortality is rather high, 6 of our 60 cases having died before the time of our publication, and doubtless others having succumbed later. No cases are reported as showing symptoms in adult life, with 1 doubtful exception. This indicates that the patients either recovered or, much more probably, died in childhood. The cause of death is usually some complication, especially bronchopneumonia.

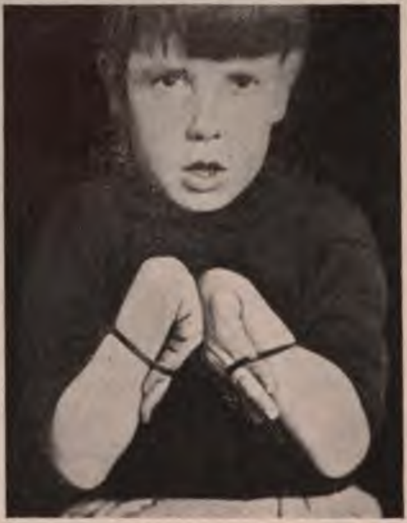

FIG. 360.—AMYOTONIA CONGENITA.

Boy of 6 years. Condition had been present from birth. Illustration shows the extreme hypermobility at the wrists. (*Charles, Brit. Jour. Child. Dis.*, 1912, *IX*, 8.)

The **diagnosis** rests upon the congenital origin, the comparatively slow course, the absence of familial disposition, and the remarkable atony of the muscles without visible atrophy. In all of these respects the malady is distinguished from the Werdnig-Hoffmann type of muscular atrophy. **Treatment** consists in the employment of electricity and massage; but has generally appeared to be of little avail.

CHAPTER VII

DISEASES OF THE PERIPHERAL NERVES

NEURITIS

In this connection will be considered certain disorders dependent upon inflammation of the nerves themselves, omitting those in which the primary cause is some disorder of the brain or spinal cord. Either one or several nerves may be involved, in the latter event usually symmetrically. The causes of neuritis are various, and the different forms, based upon the combination of cause and distribution are, therefore, best considered separately:

A. POLYNEURITIS

(Multiple Neuritis; Toxic Neuritis)

Etiology.—With the exception of the neuritis following diphtheria, which is discussed under that heading, this is a decidedly uncommon disease in early life. I cannot recall seeing over half a dozen cases. It may, however, occur in infancy. No reference is made here to the neural form of muscular atrophy (p. 390) or to the neuritic type of poliomyelitis (Vol. I, p. 531). Causes of various sorts may be operative: (1) One class of cases may be called *primary;* sometimes denominated "rheumatic," although it is questionable whether rheumatism has any real influence. Here may be included such agents as over-exertion and exposure to cold. There have been 2 cases following over-exertion under my observation. In some instances no cause whatever can be discovered. (2) *Toxic causes* are operative in a second class. Especially prominent here are arsenic, lead, and alcohol. Arsenical neuritis may follow the continued administration of this drug, especially in the treatment of chorea, and a considerable number of cases are on record. I recall at least 1 instance in my own experience. It may very occasionally occur as a result of chronic intoxication by arsenic from wall-paper, carpets, toys, and the like. Lead may also produce neuritis, the poison being derived from cakes, candies, medicines and drinking-water containing lead. Alcohol is a rare cause in childhood. The neuritis occurring in cachectic conditions is probably toxic in nature. (3) The third class of causes are *postinfectious* in nature, multiple neuritis sometimes occurring after any of the acute infectious diseases, such as scarlet fever, mumps, pertussis, malaria, measles, grippe, pneumonia, typhoid fever, and especially diphtheria. It is probably not so much the germs themselves which produce the neuritis in these cases as the toxins to which their growth has given rise. The epidemic occurrence of multiple neuritis has been reported; but it is probable that in many instances the patients were suffering from the neuritic form of poliomyelitis.

Regarding the relative frequency of the various causes, Thomas and Greenbaum[1] were able to collect from medical literature 138 cases of polyneuritis in children. Of these 48 were associated with infectious diseases, 48 were toxic, and 42 were of unknown origin.

Pathological Anatomy.—The lesions are both inflammatory and degenerative, the relative degree of each varying with the case. When inflammation predominates either the sheath of the nerve may be affected, or the interstitial connective tissue as well (*Interstitial neuritis*). In this condition the nerve is swollen and red, and there is a cellular infiltration of the sheath or between the nerve fibres. Degenerative alteration (*Parenchymatous neuritis*) is productive of disintegration of the myelin; increase of the nuclei of the sheath of Schwann; breaking up of the axis-cylinders, and finally complete destruction of the nerve-fibres, sometimes combined with increase of fibrous tissue and of fat. When this process predominates the nerves do not exhibit swelling or congestion. It is not necessary that the severer class of lesions shall develop, and many cases stop short of any serious destructive change, and complete restitution occurs.

Accompanying the neuritis there are in some cases slight alterations in the spinal cord, apparently secondary or appearing simultaneously, and consisting especially in a degree of degeneration of the ganglion cells of the anterior horns. Atrophic changes develop in the muscles supplied by the affected nerves. The degree of regeneration varies with the case and with the severity of the lesion and its duration.

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[1] Journ. Amer. Med. Assoc., 1907, XLVIII, 1397.

The involvement of the nerves as described applies more or less to forms of neuritis other than the polyneuritic type under consideration, but the changes in localized neuritis are oftener greater and more lasting.

Symptoms.—The symptoms vary greatly with the cause and the regions affected, and a classification may be followed based upon these:

1. ACUTE PRIMARY POLYNEURITIS. **Symptoms.**—The onset is sometimes rapid, the full development of the attack occurring within a few days or a week. Oftener it is more gradual, 2 to 4 weeks being required. The earliest manifestations are a symmetrical weakness and possibly pain in the extremities, especially of the muscles below the knee. Fever, headache and loss of appetite may be present or absent. There is foot-drop, with the characteristic high steppage gait attending this. The forearms may be attacked simultaneously with the legs or oftener later, and wrist-drop may develop. The muscles atrophy; the tendon-reflexes, and generally the cutaneous also, are weakened or abolished; the electrical contractility is diminished; the reaction of degeneration is often present; the sphincters are not affected; contractures may develop, and ataxia and tremor may occur. In some instances the ataxic symptoms greatly predominate over the paralytic. The paralysis is of the flaccid type and in well-marked instances there is a typical flail-like condition found on passive movement at the joints. In severe cases the paralysis becomes widespread and may even involve to some extent the muscles of respiration, and in the worst the process may attack even the cranial nerves.

Sensory disturbances consist of pain, either spontaneous or on pressure; hyperesthesia; paresthesia, or less often anesthesia. These may be largely absent, and are usually most marked early in the attack during the increase of the paralytic symptoms. The sensibility to pain, the temperature sense, and the muscle sense are all affected. Trophic disturbances are sometimes seen, such as edema, glossiness of the skin and sweating.

Prognosis and Course.—The course is moderately slow, it being often several months, sometimes a much shorter or longer time, before recovery is attained. The prognosis is usually entirely favorable. It is possible, however, for death to occur, but this is uncommon, and a fatal paralysis of deglutition, respiration, or cardiac action is generally confined to the cases dependent upon diphtheria. Persistence of any degree of atrophy or paralysis after recovery is not often observed.

2. TOXIC NEURITIS.—The description of the symptoms of polyneuritis applies also in most particulars to neuritis depending upon the action of poisons. In the arsenical cases the paralysis and pain are oftenest limited to the extremities, especially the lower; ataxia is prone to be an unusually prominent symptom; and trophic disturbances are frequent. The prognosis is usually favorable, although instances of permanent paralysis have been observed. Plumbic neuritis in children attacks the legs first and then the forearms, producing foot-drop and wrist-drop. The supinator longus and the abductors of the thumb usually escape. The prognosis is generally favorable, although death may occur from other effects of the poisoning, such as progressive cachexia or cerebral symptoms.

Alcoholic neuritis presents no special characteristics The paralysis spreads over the body as in the primary cases. Ataxia is a prominent symptom, as are sensory manifestations. The prognosis is on the whole favorable, although death may occur from the other results of chronic alcoholic intoxication frequently present.

3. Post-infectious Neuritis.—Chief in this class is the post-diphtheritic paralysis which has been considered elsewhere (Vol. I, p. 454). In this connection may be mentioned only the tendency of this diphtheritic form of polyneuritis to involve the muscles of the throat, especially the palate; the muscles of ocular accommodation; the extremities; the heart, and the respiration. The patellar reflex is almost invariably lost, no matter where the seat of the paralysis may be. Atrophy occurs as in other forms; but pain in the nerve-trunks is wanting. Other sensory disturbances may be present. The prognosis is usually good except in the event of cardiac or of severe respiratory paralysis, in both of which it is dubious and a fatal ending is frequent. Whether the cardiac involvement consists of a neuritis of the cardiac nerves or of a toxic myocarditis is not definitely determined.

Neuritis after other infectious diseases is uncommon and its symptoms do not differ materially from those of multiple neuritis in general. The paralysis is usually first in the lower extremities, especially the peroneal region, and often limited to them, or may appear in the arms also.

4. Recurrent Polyneuritis.—In this there is a decided tendency for the attack of neuritis, of the acute primary type, to recur perhaps several times. The condition is uncommon and in children especially is rare. Sereni[1] reports a case in a girl of 10 years who had 3 attacks within 3 years.

Diagnosis of Polyneuritis.—The principal diagnostic indications are the combination of sensory and motor symptoms in the same region; muscular atrophy; and altered electrical conditions, especially the reaction of degeneration. The disease may in some instances be confounded with *poliomyelitis*. The onset in the latter is, however, usually more sudden, and the general nervous symptoms more severe; the distribution is not so widespread or so symmetrical; the affection does not begin in the terminal portions of the limbs; and there is less localized tenderness and pain along the nerve-trunks. To all this, however, there are many exceptions, and cases occur in which the diagnosis is extremely difficult. This is true especially of the polyneuritic form of poliomyelitis; and it is very probable that some of the epidemic occurrences of multiple neuritis which have been reported should properly be classed as poliomyelitis. *Landry's paralysis* shows a progressive ascending advance, while polyneuritis involves parts far removed from each other, as, for instance, the upper and lower extremities while the trunk still remains unaffected. *Compression myelitis* gives pain and paralysis; but the tendon-reflexes are increased. The *muscular dystrophies* are of slower onset and sensory disturbances are not a common feature. *Neural muscular atrophy* exhibits foot-drop, as does often multiple neuritis; but there is less marked sensory disturbance in the former and the extension of the process is slow. *Infantile scurvy* has occasioned confusion on account of the pseudoparalysis and the pain which are present. The predisposition of scurvy to occur in infancy, and the development of such symptoms as swollen, painful gums; red blood-cells in the urine; and the like, are sufficient to make the diagnosis usually easy. The distinguishing of the different forms of polyneuritis from each other has been sufficiently indicated.

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[1] Il Policlinico, 1903, X, sez. med., 357.

Treatment.—The cause of the disease must first of all be determined if possible, in order to remove its further action. This is especially true of the cases of toxic neuritis. Apart from this the treatment is entirely symptomatic. The pain is to be relieved by the application of heat, or, when required, by the internal administration of bromides, antipyrine, phenacetin, or, possibly, opiates. Heat must be applied cautiously in order not to produce burns in a skin perhaps partly anesthetic. Counterirritation is not advisable. Salicylates are of no service except in so far as they relieve pain. Quinine should be employed in malarial cases. Later in the disease, when sensory symptoms have diminished and those of paralysis are well established, the employment of galvanic or faradic electricity is advisable. Still later massage may be employed. Contractures are to be guarded against by splints or bandages. In diphtheritic paralysis the element of cardiac weakness must receive first consideration, and any sudden movement of the body avoided. Full doses of strychnine, considered a favorite remedy in neuritis of the diphtheritic type and for cardiac stimulation, may be of service in other forms, and every means should be used to maintain the general nutrition.

B. LOCALIZED PERIPHERAL NEURITIS

The nerves of any portion of the body may suffer from inflammation, the result of trauma or other causes. Only those forms of localized neuritis will be considered here which may be regarded as of importance in infancy and childhood:

1. Facial Paralysis (*Bell's Palsy*). **Etiology and Pathology.**—This is one of the commoner varieties of paralysis of peripheral origin in early life. The causes are somewhat varied, all resulting in a neuritis developing somewhere in the course of the facial nerve. This may have been through trauma incurred in any way, as, for instance, during birth from the pressure of the forceps, or through other less understood methods. Surgical operations, as for the removal of cervical glands, may sometimes injure the nerve; or the presence of an inflamed parotid or lymphatic gland may occasionally produce it. A cause of importance is exposure to chilling, as by a draught of cold air, cases of this sort being often called "rheumatic." Children of any age may suffer in this way. All the causes mentioned are operative after the nerve has left the stylomastoid foramen. Within the canal it may be compressed as a result of disease of the middle-ear, especially if caries of the petrous bone is present. This is the most frequent method of production of facial paralysis in childhood and especially in infancy (Fig. 361). Within the cranium the nerve may be injured by the pressure of tumors, a basilar meningitis, or other disease of the base of the brain. The lesions found in facial paralysis are those of neuritis of any sort, as described under Polyneuritis.

Symptoms.—These vary to a certain extent with the position of the lesion. The degree of the facial paralysis due to compression by obstetrical forceps will depend upon the point where the nerve was pressed upon. Paralysis following operations for the removal of cervical glands affects only the lower branches of the nerve. "Rheumatic" neuritis and that the result of disease of the middle-ear involve, partially or completely, the whole nerve-trunk before branching occurs; and, as a result, the entire district supplied by the facial nerve is paralyzed.

In typical well-marked cases the forehead on the affected side cannot be wrinkled, the eye closed, or the edge of the nostril raised. The cheek on this side is flabby and smooth; and on efforts at smiling or at showing the teeth the mouth is drawn to the healthy side, while the other remains unmoved. Disturbance of tactile and other sensibility is absent, since the facial nerve is purely a motor one. Whatever the part of the nerve affected, from the periphery up to the nucleus in the pons inclusive, there are found the electrical reactions characteristic of neuritis. There is, namely, great or complete loss of the faradic contractility, and soon of the galvanic response as well; while later both may return, the latter then showing the reaction of degeneration. Lesions above the nucleus are not productive of these changes.

Course and Prognosis.—In the paralysis dependent upon birth-lesions the course is rather short and the prognosis favorable, recovery generally being complete in from 1 to 2 weeks; although more serious cases sometimes continue longer or exceptionally persist permanently. The instances due to exposure to cold are somewhat slower in convalescing, but recovery is the rule after a few weeks or months. When the neuritis follows middle-ear disease, the course is longer and liable to periods of temporary improvement followed by remissions; and the ultimate recovery is much more uncertain. The prognosis is unfavorable when the disease depends upon lesions within the cranium. Recovery is often very slow after injury by operations on the face or neck, but generally is finally complete, if the injury has not been too severe.

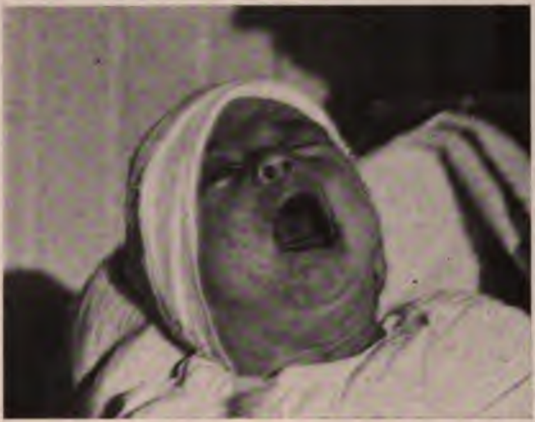

FIG. 361.—FACIAL PARALYSIS.

Case in an infant of 3 months in the Children's Ward of the University Hospital, Philadelphia. *Courtesy of Dr. J. C. Gittings.* (*See also, Arch. of Pediat.*, 1908, *XXV*, 446.)

Diagnosis.—This is usually easy in cases at all typical. Paralysis in all the branches of the facial nerve indicates a peripheral lesion; while only the lower branches may be peripherally affected after operations on the neck, or involved centrally by a lesion above the pontine nucleus. In the latter case, however, there is no reaction of degeneration, and if the lesion is at the base of the brain other cranial nerves, especially the auditory, are liable to be affected as well. When the lesion is in the pons the paralysis of the face is usually complete, and paralysis of the body of a hemiplegic type is usually present also. The diagnosis of the localization of the lesion lower in the course of the nerve is often difficult, and sometimes not to be made with certainty. Lesions below the stylomastoid foramen have no attending disturbances of taste or of salivary secretion or hearing. This is true also when the nerve is diseased within the canal below the departure of the chorda tympani but where in addition the posterior auricular branch of the nerve is involved. If the lesion is in the region of the chorda tympani, there is disturbance of taste and diminution of the secretion of saliva. If the portion above this, but below the geniculate ganglion, is affected, there is added to these changes abnormal sharpness of hearing. A lesion of the geniculate ganglion shows besides these same symptoms disturbance of lachrymation and perhaps paralysis of the palate.

Not to be forgotten is the possibility of the occurrence of facial palsy as one of the symptoms of poliomyelitis (Vol. I, p. 530, Fig. 169). The characteristic paralysis in some of the limbs is, however, generally present, or there may have been a history of its occurrence, and this will serve to distinguish from facial neuritis. I have, however, seen facial paralysis practically the only symptom remaining after poliomyelitis.

Treatment.—That of the cause is of first importance. When this is entirely cerebral little can be done. When disease of the ear is at fault, prompt treatment for this condition is indicated; although it by no means follows that relief of the facial palsy will result. In cases depending upon exposure to cold, the iodides may be given, although the majority of cases would recover equally well if let alone. A weak galvanic current may be applied, and the placing of a small blister over the exit of the nerve is also recommended, but the advisability of this is questionable.

2. Obstetrical Paralysis (*Neuritis of the Brachial Plexus; Erb's Paralysis*. **Etiology.**—By this term is designated the partial paralysis of the arm sometimes produced during birth. It is oftenest associated with difficult and prolonged labors. An analysis by Stransky[1] of 94 cases showed 50 in which labor had to be manually assisted, and only 2 of spontaneous birth; 37 were vertex presentations and 19 breech or foot presentations. In cases of vertex presentation pressure of the forceps on the nerves issuing from the neck, the employment of a traction hook in the axilla, or the use of the physicians finger in the same position, may injure the brachial plexus, although the injury may occur without these procedures. Allowing for the much greater frequency of vertex presentation, relatively the large number of cases of obstetrical paralysis occur in breech presentations, and may then be the result of pressure above the clavicles in efforts at delivery of the head, or traction used in bringing down the arms if above it. The opinion has been entertained by many, and supported especially by Thomas,[2] that in the majority of cases paralysis is not caused so much by a direct trauma of the brachial plexus as primarily by a posterior subluxation of the shoulder-joint, with subsequent involvement of the plexus in the resulting axillary inflammation. On the other hand, an analysis by Sever[3] of 470 collected cases led him to the support of the etiological influence of primary injury of the plexus.

Pathological Anatomy.—The lesions consist in the stretching of some of the nerve-fibres in the milder cases, or of actual rupture of large nerve-trunks in the severer ones. The nerve-sheath may be injured, and there may be extravasation of blood into it, or more widely spread with consecutive inflammation. In severe cases there may be an inflammatory process involving all the nerves of the plexus in fibrous tissue.

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[1] Centralbl. f. d. Grenzgeb. der Med. u. Chir., 1902, V, 669.

[2] Annals of Surgery, 1914, Feb.

[3] Amer. Jour. Dis. Child., 1916, XII, 541.

Symptoms.—These vary with the degree of the injury. As a rule, the paralysis is unilateral, but bilateral paralysis is exceptionally seen. Oftenest the upper portion of the brachial plexus upon one side is involved (*Erb's paralysis*)[1] with the result that the deltoid, supraspinatus, brachialis anticus, biceps and supinator longus are paralyzed to a varying degree. In Stransky's 94 cases, 80 per cent. were of the Erb type. In the milder instances the condition may not be discovered for some weeks, but in those more severe it is evident immediately or soon after birth. The paralysis is of the flaccid type, and in well-marked cases the arm hangs helpless at the side (Fig. 362). It is rotated inward, the forearm pronated, and the palm of the hand directed outward. The forearm cannot be voluntarily flexed at the elbow, but if flexed by passive movement can be extended by the patient. The movements of the muscles of the forearm and of the fingers are unaffected. There is little if any pain, and disturbances of sensation of any sort are slight, and limited to moderate anesthesia of the shoulder and the outer side of the upper arm. The extremity is colder than normal. Atrophy of the muscles soon develops, although not readily discovered early in the case. The electrical contractility is diminished, and in severe cases the reaction of degeneration can be obtained.

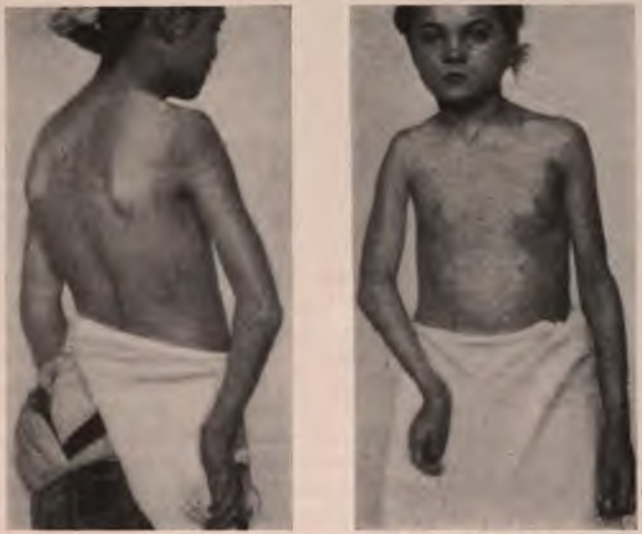

FIG. 362.—OBSTETRIC PARALYSIS, UPPER ARM TYPE.

Characteristic position of the arm. (*Bullard, Amer. Jour. Med. Sci.*, 1907, *CXXXIV*, 99.)

In the much less frequent instances where only the lower portion of the brachial plexus has suffered, the upper arm is normal, but the hand and forearm are paralyzed (*Klumpke's paralysis*).[2] When the whole plexus is involved, a still less frequent condition, the forearm and hand share the paralysis of the upper arm. All degrees of paralysis are present in lesions of the plexus, from slight weakness up to complete helplessness. Fracture of the clavicle or the humerus, or separation of the epiphysis, may be seen as complications, and dislocation of the shoulder-joint is common.

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[1] Ziemsson's Handb. d. spec. Path. u. Therap., 1874, XII, 1, 492.

[2] Rev. de méd., 1885, V, 591.

Course and Prognosis.—The duration is variable, and the prognosis uncertain, although in general favorable. The mildest cases may recover completely in a few weeks, but most often 2 or 3 months are required before this occurs. In still other cases some degree of paralysis remains always, or there may be no improvement whatever. The chances for recovery depend upon the severity of the lesion, and may be based to a certain extent upon the electrical reactions. When faradic contractility has never been entirely lost recovery is rapid; but if both galvanic and faradic contractility disappear, or the reaction of degeneration is present, recovery may be very slow, and is always uncertain. As time passes in severe cases the growth of the limb is retarded and contractures are liable to develop.

Diagnosis.—This is usually easy, if the case is seen early. It rests upon the production of the condition at birth; the usual location of the paralysis in the upper arm; its flaccid character; and the diminished electrical contraction, perhaps with the reaction of degeneration. Examination of the shoulder may show luxation of the humerus. Brachial monoplegia dependent upon an intracranial lesion is of very rare occurrence. There is in it no diminution of faradic contractility, and the spastic character of the paralysis becomes evident as time passes. An obstetrical paralysis which is seen late in the disease, after the patient is a year or more old, may be readily confounded with one of the arm persisting after poliomyelitis, since both diseases have atrophic paralysis of a flaccid type. The history of the case will lead to a correct diagnosis, since the age of onset is different, the degree of wasting is usually greater in obstetrical paralysis and the localization is characteristic. Syphilitic epiphysitis and infantile scurvy have a later age of development, and there is no true paralysis present. Fractures occurring at birth are painful, and this leads to an examination which reveals the nature of the trouble.

Treatment.—No treatment at all should be employed for at least 2 weeks following the occurrence of the lesion. After this time, if a rapid improvement is not beginning, systematic procedures should be instituted. These consist in gentle massage, passive movements, and the employment of electricity; choosing the faradic current if contractions are obtained with it, otherwise the galvanic current. The treatments should be of short duration, but be given daily and continued over a long period. Dislocation of the shoulder should be sought for and reduced if present. If contractures begin to develop the affected region should be kept in splints, except when treatment is being given. In cases which are protracted, and show little or no response to the means employed, surgical intervention may be invoked, consisting of a dissection of the different nerve-trunks of the plexus from the masses of fibrous tissue in which they are embedded. The results, however, are not always satisfactory.

3. Other Peripheral Paralyses.—As already stated localized peripheral neuritis may occur in almost any of the nerves. Very exceptionally there may be a birth palsy of the lumbar and sacral plexus, produced by obstetrical manipulations, such as forcible traction upon the lower extremities. The condition is to be distinguished from a hematomyelia (p. 377) and subsequent myelitis produced in the same way. The presence of complete anesthesia observed in the latter condition is a distinguishing feature. At a later period of life the peroneal nerve may suffer through injury to the legs, or the crural nerve be affected by pressure of a psoas abscess or of an appendicitis. The nerves of the forearm may be paralyzed by trauma, or those of the upper arm damaged by dislocation of the humerus, fractures, or in other ways. The various cranial nerves may suffer from neuritis, in addition to the facial as already described. This may depend upon a nuclear lesion, or be the result of pressure by a tumor or a meningitis. The subject is appropriately discussed more fully in treatises upon nervous diseases.

4. Facial Hemiatrophy.—This disease, rare at any time of life, often begins in childhood. Its nature is not clearly understood, but it may be referred to here on the ground that it has been supposed to depend upon an inflammation of the trigeminal nerve and of the Gasserian ganglion. It has been known to develop after trauma of the face, or to follow infectious diseases. The symptoms consist of a progressive wasting of the tissues of one side of the face, including the skin, subcutaneous tissue, muscles, and bones. Sometimes both sides of the face are affected. The process begins in some one spot, usually in the cheek, which grows thin and sometimes white or discolored; and gradually the whole side may waste and become much smaller than the other (Fig. 363). The electrical reactions and the muscular movements are normal. Neuralgic pain may occur early in the disease.

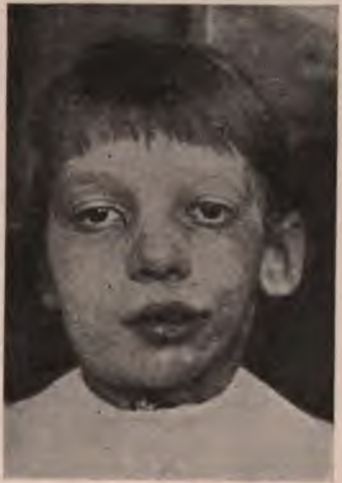

FIG. 363.—FACIAL HEMIATROPHY. Girl of 12 years. The symptoms were first noted 2 years previously. (*Ashby, Monatsschr. f. Kinderh.*, 1903, *II*, 64.)

MORBID GROWTHS OF THE PERIPHERAL NERVES

Primary neoplasms of the nerves are a rare occurrence in early life, and the same is true of involvement secondary to morbid growths developing elsewhere. One form only is of some importance: *General neurofibromatosis*, or von Recklinghausen's Disease,[1] and even this is but seldom seen. It is sometimes hereditary or familial, and has been observed in early childhood or even at birth. The symptoms consist in the development of numerous cutaneous tumors; small pigmented spots in the skin; and tumors of any of the cranial, spinal, or sympathetic nerve-trunks, or of the spinal nerve-roots. The growths may be of pin-head size, or be much larger and occupy a nerve-plexus. When on the spinal nerve-trunks they are generally insensitive, sometimes pedunculated, often very numerous, and frequently produce no clinical manifestations whatever. When situated on cranial nerves or the spinal nerve-roots they give rise to varied and severe symptoms which suggest morbid growths of the spinal cord or of the brain, and the disease may lead to a fatal ending. In other cases less severe the condition may long remain stationary but does not disappear. The diagnosis is easy, based on the triad of symptoms mentioned. Treatment is unavailing.

[1] Ueber Multiplen Fibrome der Haut und ihre Beziehung zu den Multiplen Neuromen, Berlin, 1882.

SECTION IX

DISEASES OF THE MUSCLES, BONES AND JOINTS

CHAPTER I

DISEASES OF THE MUSCLES

The most important of these, the muscular dystrophies, as well as amyotonia congenita, have already been described under Diseases of the Nervous System for reasons there explained. There remain a few to which some attention must be given.

MALFORMATIONS OF THE MUSCLES

These are occasionally seen, and are very varied in nature. They have been carefully studied by Bing.[1] The most common is congenital **absence of the pectoral muscle,** oftenest of the lower part upon one side. Of this malformation Bing reported 102 cases. Less frequently there may be a **defect** in the trapezius, serratus, quadriceps femoris, or other muscles. Characteristic changes are observed in the case of the pectoral malformation, the anterior portion of the upper chest-wall being much flattened. Other malformations are frequently combined with this, such as imperfect development of the breast; abnormally high position of the shoulders (Sprengel's deformity); malformations of the hand, etc. Sometimes a number of different malformations of the muscles are encountered in the same individual. There are usually few if any symptoms evident, the patient learning to use other muscles to replace the action of the defective ones.

Congenital **absence of the abdominal muscles** is a rare anomaly. In this the abdominal wall of the affected region is very thin and the skin wrinkled. Coughing and crying are interfered with, and death is very likely to take place from bronchitis. The few reported cases, up to less than 10 in number, have been collected by Garrod and Badies.[2]

MYOSITIS

Of this disorder several varieties may be noted. As a local process a *suppurating myositis* in one or more muscles may occur as a result of trauma or through pyogenic infection acquired in other ways. A *non-suppurating localized myositis* is represented by muscular rheumatism; is well illustrated in some cases of torticollis; or may be the result of trauma or follow some of the infectious diseases. A multiple suppurating myositis may occur as an unusual manifestation of general sepsis.

More interesting are the varieties of polymyositis of a non-suppurating type, which may be grouped for convenience under (1) Simple polymyositis, (2) Progressive myositis ossificans.

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[1] Virchow's Archiv, 1902, CLXX, 175.

[2] Med.-Chir. Transac., 1905, LXXXVIII, 363.

1. PRIMARY SIMPLE POLYMYOSITIS

Etiology and Pathological Anatomy.—This is a rare affection, perhaps more common in childhood than later, of which there are several sub-varieties, known as myositis fibrosa, dermatomyositis, etc. The cause is entirely unknown. Any period of life may be affected. The lesions are limited to the muscles. These are stiff and firm, and on section are tough, whitish and grating. There is edematous and small-celled infiltration, and great increase of the interstitial connective tissue, with a secondary degeneration of the muscle-fibres. Inflammatory edema of the skin may also be present. When this latter condition predominates, the term *dermatomyositis* is employed; if a hemorrhagic exudate into the skin or mucous membrane occurs, *hemorrhagic polymyositis* is spoken of; and to the more chronic cases with a predominance of foci of hyperplasia of the interstitial connective tissue, the term *myositis fibrosa* has been applied.

Symptoms.—These begin acutely in *dermatomyositis*, with such characterisitc manifestations as fever, loss of appetite, malaise, headache, enlargement of the spleen, and, very frequently, gastrointestinal symptoms. Then, with the fever and other features persisting, the extremities, usually first the lower, become painful on touch and movement, and the muscles swollen and hard; and later other muscles of the body including those of the face are affected. The patient finally loses the power to move, and is confined, helpless and stiff, to bed. Cotemporaneously with the symptoms described, or sometimes later, there develops a widespread, tense, inflammatory cutaneous edema, not readily pitted, appearing on the surface as an erythema of different sorts, and involving the whole body including the face. The edema is often so marked that the condition of the muscle cannot at first be determined.

The *fibrous form* is without any acute general symptoms, either initial or later, and the cutaneous edema is absent. The course is slower and more chronic than in the type previously described, the condition less often becomes so widespread over the body, and there is less pain.

Course and Prognosis.—In the severer cases the extension of the trouble is rapid; the functions of the larnyx, swallowing, respiration, and cardiac action are interfered with, and death occurs in from 1 to 8 weeks. In other instances the acute symptoms are absent or subside, and the condition passes into a subacute or chronic form, lasting 3 to 6 months or even several years. Periods of remission are often seen, followed by exacerbations of the general symptoms and renewed extension of the disease; favorable cases finally recovering, although with the muscles perhaps atrophied. The prognosis is always serious, more than half of the cases ending fatally; although better when the disease develops in childhood, and decidedly more favorable in the fibrous form..

Diagnosis.—This is to be made chiefly from trichinous myositis, which resembles the form described so exactly that a family history of the disease and the excision of a portion of muscle constitute the only means of differentiation. Acute rheumatism exhibits involvement of the joints. Myositis ossificans is characterized by the bony deposits.

Treatment.—This is entirely symptomatic and consists in complete rest in the acute stages, with measures to relieve the pain. Later gentle massage and electricity can be tried.

2. MYOSITIS OSSIFICANS

Etiology and Pathological Anatomy.—This uncommon affection is one of early life, 27 of Lorenz's[1] 51 collected cases having commenced in the first 5 years, 11 from 5 to 15 years, and some being even congenital. The cause is unknown, but probably is some congenital constitutional disturbance, as indicated by the combination with the muscular lesions of the deformity of the fingers and toes which is sometimes seen. A local trauma is undoubtedly the occasion of the development of the condition in a certain region; but is in no way the primary etiological factor. The majority of the cases occur in males. The lesions consist at first of foci of cellular infiltration similar to the process seen in simple myositis; later of connective-tissue overgrowth as in the fibrous form. These finally advance to the production of nodules of true bone, situated primarily in the muscles, but later attaching themselves to some extent to the bones of the body; or sometimes developing primarily here also. A localized fibrous myositis exists in the vicinity of the bony masses.

Symptoms.—These show themselves first in the muscles of the neck and back, but eventually spread to other regions, although those first mentioned exhibit finally the greatest number of nodules. With fever and pain there develops a localized and sometimes tender swelling in the muscles, with edema in the surrounding parts. In a few days the fever and evidences of inflammation subside, and the swelling may disappear completely; to become permanent only after several recurrences in the same locality have taken place. In other cases the primary swelling does not disappear, but remains as a hard nodule; and then gradually, perhaps after more relapses in the same locality, becomes still harder and finally actually bony. There occur repeated attacks of this nature, often after some slight localized trauma, with development of fresh nodules; until in advanced cases a large majority of the muscles are involved, including finally the masseters and the temporals. The bony masses, once formed, are not tender; are at first of pea-size; but gradually grow and many of them coalesce into large, irregular masses. They are at the beginning movable under the skin, but are finally fixed through attachment to the bones.

At first there are no subjective symptoms produced by the bony masses; but their increase in number and size, their attachment to the skeleton, and the accompanying fibrous myositis, result in an advancing interference with muscular activity, until the joints become stiff and the patient rigid and helpless. The involvement of the masseters interferes greatly with opening the mouth and with mastication.

Course and Prognosis.—The course of the disease is very prolonged, the patient always growing worse, although always with long temporary pauses followed by exacerbations and extensions. It may be long, too, before the fibrous induration composing a nodule becomes true bone; but it is possible for the bony change to develop speedily. The disease usually lasts over many years, but sometimes runs a more rapid course. Microdactylia is a very common complication, the thumb and big-toe being smaller than normal.

The **diagnosis** is easy after the formation of bone has taken place. Multiple exostoses are attached to the bone from the very beginning, and cause no widespread interference with the movements of the body. **Treatment** is entirely unavailing. It is important to guard against even slight traumata as far as possible.

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[1] Nothnagel's Handb. der spec. Path. u. Therap., 1904, XI, 3, 1, 273.

TORTICOLLIS

(Wry Neck)

In some instances purely a muscular lesion, in others a myalgia, in still others an expression of a nervous disease, torticollis may properly be considered as on the border line as regards classification.

Etiology.—The condition is a common one at any time of life and may be due to a number of varied causes. Much the most frequent of these is muscular rheumatism;—a rheumatic myositis or fibrositis of short duration (see Vol. I, p. 626), oftenest the result of exposure to cold. An analogous condition may be encountered in other muscle-groups, being represented, for instance, by lumbago. Less often torticollis may be brought about by caries of the cervical vertebræ; or by pressure of an inflamed cervical gland or of an abscess or tumor in the neck upon the spinal accessory nerve; or be the result of some primary affection of the nerve itself. Cases dating from birth or seen soon after it may be dependent upon a congenital anomaly such as a shortening of the muscles, or be the sequel remaining after a hematoma of the muscles of the neck produced by a trauma received during birth. It is a matter of dispute in these early cases whether congenital anomaly or trauma predominates etiologically. It is evident that some of the causes have no relationship at all to the nervous system, while others act directly or indirectly by engendering a spasm in the muscles supplied chiefly by the spinal accessory nerve. A spasm here may be brought about by influences acting anywhere upon the nerve throughout its course, from the cerebral cortex to the terminal branches. Other muscles of the neck besides those innervated by this nerve may also be affected. Wry neck of the purely spasmodic form, dependent upon a primary spinal-accessory affection, is of very uncommon occurrence in early life. Very exceptionally a torticollis may be the result of paralysis of the nerve of the healthy side, allowing the head to be gradually drawn to the other.

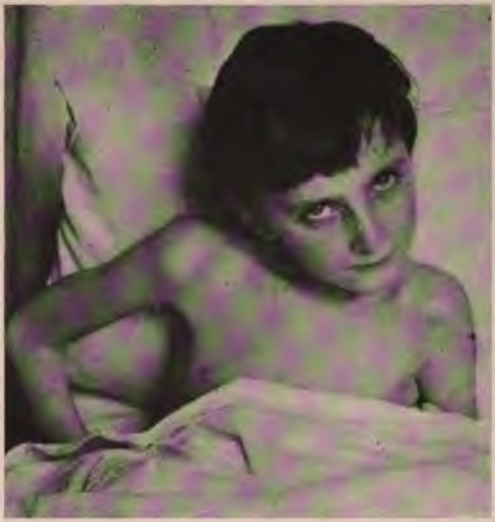

Fig. 364.—Rheumatic Torticollis.

From a patient in the Children's Ward of the Hospital of the University of Pennsylvania.

Symptoms.—The disease may be acute or chronic, congenital or acquired. The congenital cases, or those dependent upon trauma at birth, both of them uncommon, are chronic in their course; the rheumatic cases are oftener acute; those due to other causes persist while the acting cause remains. The *congenital* condition is not infrequently combined with other deformities, among them facial asymmetry. The alteration in the position of the head is generally not discovered for some time after birth. In the typically *acquired* cases the disease is situated chiefly in the sternocleidomastoid muscle upon one side, as a result of which the muscle stands out as a rigid, prominent cord, and the head is drawn to the affected side and downward toward the thorax, with the chin tilted upward and toward the sound side (Fig. 364). Very frequently the trapezius is also involved. This draws the head somewhat backward on the affected side, and increases the tilting of the chin mentioned. The degree of distortion and its exact nature depend upon the degree to which the different muscles are affected. Sometimes the posterior muscle-groups on both sides are the seat of the disease, in which event the head is pulled backward, the chin slightly elevated, and turning the head to either side is interfered with. The amount of pain and tenderness existent also varies with the case. Pain may be constantly present and severe, or develop only when efforts are made to move the head. Pain and tenderness are especially marked in the acute rheumatic cases.

Course and Prognosis.—The duration of the acute cases is generally short and recovery takes place in a few days, or at the most a few weeks. It is not uncommon, however, for an attack beginning acutely, and apparently of the rheumatic type, to last a longer time, and it is even possible for a chronic myositis to develop and the course to be very much prolonged. When torticollis is due to pressure upon or other involvement of the spinal accessory nerve, the duration will depend upon the possibility of the removal of the lesion. When the cause is a cervical caries the prognosis is unfavorable, unless the treatment of the primary disorder is successful. The congenital cases last indefinitely, although the milder ones may be relieved by appropriate treatment. In very many instances of torticollis a decided tendency to recurrence is observed; and many others, if not subsiding promptly, show a disposition to become chronic. Any case which has lasted a considerable time has the prognosis made more unfavorable by the likelihood of some permanent alteration of the muscle developing, with a persistent shortening of it.

Diagnosis.—The recognition of a deviation of the position of the neck is easy, but the determining of the etiological factor is not always as simple. The various possible causes should be considered as applied to the individual case. In the rheumatic cases the tenderness and pain in the affected region are generally especially well marked. Whenever the patient holds the head stiffly in the median position, the existence of a cervical caries should be carefully eliminated before the idea of a simple affection of the muscles is entertained. In caries the rigidity is usually greater, as is the pain on any attempted movement.

Treatment.—The determining of the cause is naturally followed by the attempt to remove this if possible. In the ordinary acute rheumatic form of the disease relief is obtained by the administration of the salicylates, often advantageously combined with phenacetin; the protection of the neck from cold; the employment of a hot-water bag, and the application of such counterirritants as mustard or capsicum in some form. All the congenital cases, and others which have lasted some weeks, require efforts at correction and support by orthopedic apparatus. This may be of avail in the milder instances if commenced sufficiently early. Massage and electricity may also be tried. Yet after the condition has continued several months operative procedures are often necessary. The results have not been encouraging once the deformity has become permanently established.

MYASTHENIA GRAVIS

(Asthenic Bulbar Paralysis)

The exact nature and relationships of this disease are not determined, but it may conveniently be considered in this connection. It is a very rare affection in childhood, although a few cases are on record (Palmer).[1] The cause is unknown, and the pathological lesions are not well understood. The only changes which have been observed consist in the occurrence of masses of small round-cells in the muscles; but these have been discovered in other diseases as well. An enlargement of the thymus gland has been sometimes noticed. Nothing has been found wrong with the nervous system. The disorder may be the result of the action upon the muscles of some toxic metabolic product; or may be a congenital constitutional anomaly.

The **symptoms** consist in a state of excessive fatigue and ready exhaustion, seen first and especially in the muscles controlling the movements of the eyes, swallowing, speech, and mastication; and extending to those of the neck, trunk and the extremities. Movements of these muscles are possible only for a few moments at a time; then after a short rest the power returns for a similarly brief period. There is no muscular atrophy, and sensation is undisturbed. The electrical excitability is normal but soon exhausted; to reappear after a short rest. The onset is rather slow; the course chronic, often with periods of temporary improvement. The prognosis is unfavorable; death usually occurring after some months or years of illness, often from respiratory involvement, interference with swallowing, or inanition. Recovery, however, may take place. The diagnosis is often difficult. It is based chiefly upon the distribution of the asthenia; the rapid exhaustion with rapid temporary regaining of power, and the absence of atrophy. Treatment is of little avail, although absolute rest and the influence of tonic remedies may be tried.

MYOTONIA CONGENITA

(Thomsen's Disease)

Although cases had been reported previously, this rare condition was brought into prominence by the writings of Thomsen in 1876.[2] It is usually a congenital disorder, but oftenest not recognized until school life or adolescence; and is of a typically familial and hereditary nature, frequently extending through several generations. Males are more frequently affected than females. There are no characteristic lesions known, the only changes discovered being enlargement of and granular deposit in the muscle-fibres, but no evidence of degeneration.

[1] Guys Hospital Reports, 1908, LXII, 64.

[2] Arch. f. Psychiat. u. Nervenkr., 1876, VI, 702

Symptoms.—The characteristic symptoms of the well-developed cases consist in a temporary rigidity of the muscles when efforts are first made to put them into action. This is oftenest experienced in the legs. The patient on starting to walk is able to do so only stiffly and slowly; but after a few steps the stiffness disappears and he can walk entirely normally. If grasping any object, such as the hand of another person, he is unable to let go of it for several seconds. Less often the muscles of other parts of the body are involved. Thus sucking has been reported interfered with in cases showing symptoms in infancy; or there may be an involuntary pause at the beginning of an effort at crying. In older subjects the first efforts at mastication, talking, or at opening the eyes, may be rendered difficult by the slow and stiff action of the muscles. The difficulty with all these movements disappears promptly if effort is persisted in, and does not return until a renewed attempt at movement is made after a period of rest. The distribution of the malady is symmetrical, although it may perhaps be more marked on one side of the body. The condition is made worse by exposure to cold, or by excitement or the consciousness of being observed. There are no sensory disturbances, and the muscles are well developed or abnormally large. Tapping of a muscle reveals an increase of mechanical excitability, there being produced a slow contraction and increased prominence of the muscle, with a central depression which disappears slowly after some seconds. The muscles also exhibit an unduly active but slow response to both the faradic and the galvanic current of weak strength (Myotonic Reaction of Erb)[1] while strong currents produce a series of small wave-like contractions.

The **course** of the disease is prolonged, lasting through life, although sometimes with periods of temporary improvement. It does not affect the duration of life, but in severe cases may interfere greatly with occupational activity.

Diagnosis.—This is easily made; based upon the characteristic stiffness of movement and the electrical and mechanical reactions of the muscles. Mere slowness of movement is not sufficient, since this may be observed in other conditions. Tetany is distinguished by the character of the contractions and the difference in the electrical reactions. (See Tetany, p. 253.) There is absolutely no resemblance to myatonia congenita (amyotonia congenita) except in the close and confusing similarity of titles. **Treatment** is without promise. Persistent employment of massage and of active and passive muscular movements has been recommended.

PARAMYOCLONUS MULTIPLEX

(Myoclonia)

This uncommon condition, described by Friedreich,[2] may occur at any period of life, but is rare in childhood. It might with propriety be classed among the spasmodic nervous affections. It occurs principally in males and is sometimes hereditary or familial. Over-excitement or over-exertion operates as a direct cause. The disease resembles somewhat, but is distinct from, electric chorea, convulsive tic, and hysteria. The **symptoms** consist in very rapidly repeated, rhythmic, clonic contractions in many different muscles of the body, as a rule symmetrical in distribution. The extremities are oftenest involved, while the face usually escapes. The excursus of the movements is generally slight, but in some severe cases the action may be violent. They come on in paroxysmal attacks and are entirely beyond the control of the patient, or may be constantly present except during sleep. Tremor may occur between the attacks. There are no sensory, psychic, or electrical disturbances. The **prognosis** as to recovery is very uncertain; although some cases of cessation of the disease have been reported. Treatment is without effect.

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[1] Erb, Die Thomsensche Krankheit, 1886.

[2] Virchow's Archiv, 1881, LXXXVI, 421.

PERIODICAL PARALYSIS

This strange and uncommon malady exhibits a very distinct hereditary and familial occurrence, being inherited through several generations. Of 53 reported cases collected by E. W. Taylor,[1] 35 occurred in 3 families. Cases have been seen in childhood, but the disease usually develops at the age of puberty or adolescence. The youngest of 64 cases collected by Odda and Audibert[2] was in a child of 5 years, but Buzzard[3] reported 2 instances in one family which developed as soon as the children "were first able to move about." Over-exertion, unusual rest, or a too hearty meal would appear to bring on individual attacks, but generally no cause is discoverable.

The **pathology** is unknown, but it would seem probable that an autointoxication occurs, which temporarily affects the muscles until they are relieved by the elimination of the poison. Portions of excised muscles have shown no changes sufficient to explain the condition. The **symptoms** consist in attacks of flaccid paralysis of varying degree, developing often during sleep, and affecting generally first the lower limbs, but in the course of a few hours, or a much shorter time, extending to the upper extremities and the trunk. The muscles supplied by the cranial nerves usually escape, although ptosis and difficulty in swallowing have been observed. Sensation and the mental condition are unaffected; the reflexes are abolished; the electrical response is greatly diminished or even entirely absent to the strongest currents. Excretion of urine is often interfered with by the paralysis of the abdominal muscles. Profuse perspiration may occur, and sometimes signs of cardiac dilatation. The duration of an attack is variable; the symptoms gradually disappearing and recovery being complete in from a few hours up to 2 or 3 days. The frequency of the recurrence also varies, ranging from daily up to every few weeks, although sometimes there may be intervals of months. The disease generally ceases after the age of 50 years, but has occasionally been directly fatal. The **diagnosis** offers little difficulty after the first attack. The peculiar lack of electrical response excludes hysteria. On the theory that a toxic state is present, alkaline diuretics have been recommended in the line of **treatment,** with apparent benefit. In the way of prevention, over-excitement is to be avoided and the diet watched carefully.

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[1] Jour. of Nerv. and Mental Dis., 1898, XXV, 637.
[2] Arch. gén. de méd., 1902, CLXXXIX, 290.
[3] Lancet, 1901, II, 1564.

CHAPTER II

DISEASES OF THE BONES AND JOINTS

Although a subject largely surgical, there are yet certain topics which demand the attention of every physician and require some consideration, or at least brief mention, here. It is the physician, rather than the surgeon, who is the first to encounter the diseased condition, and upon whom a provisional diagnosis depends.

MALFORMATIONS OF THE BONES

These are varied and numerous. Many are congenital in origin; others acquired. Some are of sufficient importance to warrant independent consideration. Others, also of importance, are described in studying the symptoms of the primary diseases of which the deformities constitute but a part.

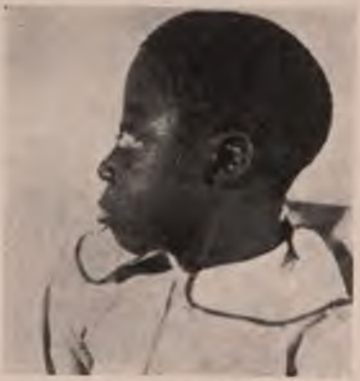

Fig. 365.—Acrocephaly.

Colored boy of 6 years. Dome-shaped skull and exophthalmos. (*Lewin, Amer. Jour. Dis. Child.*, 1917, *XIII*, 68.)

The head may be congenitally deformed in various ways, or develop an acquired deformity. The alterations of the shape depending upon rachitis and hydrocephalus; those attending certain forms of idiocy; the tumors of chloroma and malignant hypernephroma; the hyperostosis of syphilis and the malformations of cleidocranial dysostosis are referred to under other topics. Brief reference may be made to the curious congenital malformation denominated **oxycephaly,** acrocephaly or tower-skull; in which the vault is dome-shaped (Fig. 365). Other deformities are liable to accompany this. Exophthalmos is generally present, and nystagmus and paralysis of some of the ocular muscles are often attendant. The mental state may be normal or defective. The condition has been reviewed by Lewin.[1]

The bones of the extremities, together with the accompanying soft parts, may be absent or imperfectly developed. As a result are produced such malformations as **ectromelia,** where one or more limbs are entirely absent; **hemimelia,** where the distal portion is absent, and **phocomelia,** where the proximal part is missing, the remaining portion arising directly from the trunk. The absence or defect of one of the bones of the forearm or of the leg results in deformity at the wrist, ankle or knee. **Club-hand** (Fig. 366) may be produced in this way. Among defects of the hands and feet are especially to be mentioned **polydactylism** and **syndactylism.** Supernumerary fingers or toes (Fig. 367) is a defornity often hereditary and not infrequently combined with other malformations.

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[1] Amer. Journ. Dis. Child., 1917, XIII, 61.

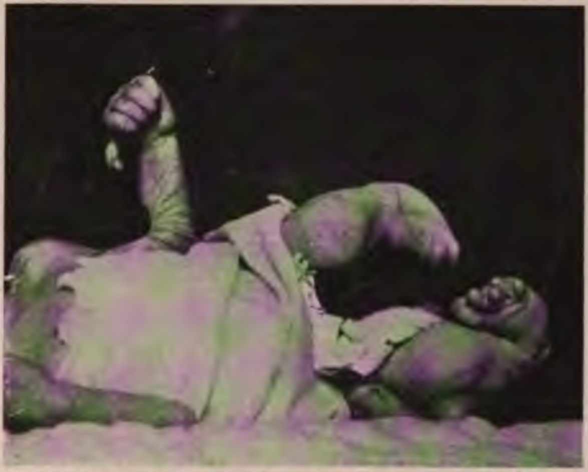

FIG. 366.—NEWBORN INFANT SHOWING CLUB-HAND AND CLUB-FOOT.
Courtesy of Dr. John D. Target.

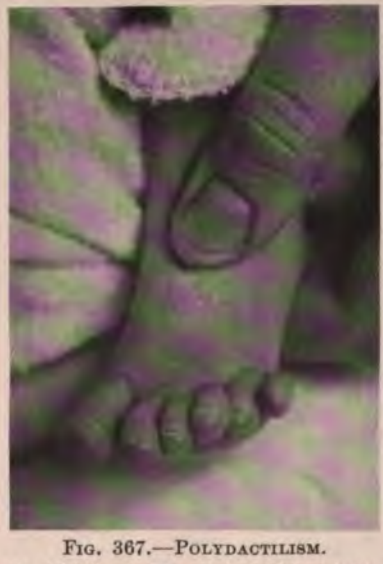

FIG. 367.—POLYDACTILISM.

Infant of 5 weeks in the Children's Hospital of Philadelphia. 6 fingers on each hand, and 6 toes on each foot, nystagmus.

The condition is frequently a readily operable one. Syndactylism (Fig. 368) may vary from a simple webbing of some of the fingers to a much more complete fusion. Operative treatment is important to render the hands properly useful. **Hypertrophy of the fingers and toes** (Fig. 369) is occasionally observed; sometimes the soft parts alone being involved, sometimes the bony tissue as well. In addition to these faults of development there are deformities from prenatal accident, such as **amputation** or other injury of a limb by amniotic bands, or through strangulation by the umbilical cord.

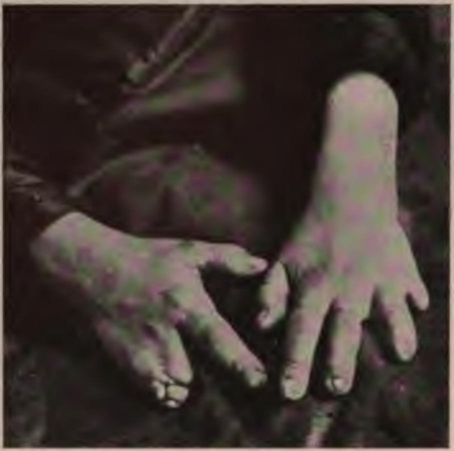

Fig. 368.—Syndactylism.

Slight in the left hand; extensive in the right. (*Courtesy of Dr. H. R. Wharton.*)

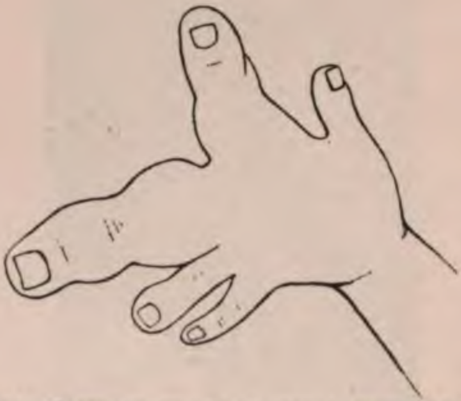

Fig. 369.—Congenital Hypertrophy with Lateral Distortion of the Index and Middle Fingers.

(*Annandale, Malformations, Diseases, and Injuries of the Fingers and Toes*, 1866, *Pl. I, Fig.* 1.)

CONGENITAL DISLOCATION OF THE HIP-JOINT

This is encountered in less than 0.5 per cent. of births (Willard),[1] oftenest in females in the proportion of about 7:1; and is bilateral in about 40 per cent. of the cases. It is often hereditary and combined with other congenital malformations. The upper border of the acetabulum is defective, and as a result the head of the femur is gradually displaced as the child grows older, although still within the capsule, and rests finally upon the dorsum of the ilium.

Symptoms.—The characteristic symptoms are generally first observed when the child begins to walk. In the case of unilateral dislocation there then develops a decided limp; the trochanter can be found above Nélaton's line; there is moderate lordosis; the thigh is shorter than normal and the natofemoral fold higher; the head of the bone is absent from the normal position in the groin to the outer side of the femoral vessels; and when the child is standing upon the dislocated leg and elevating the other the pelvis is inclined downward toward the healthy side (Trendelenburg's symptom). In the case of double dislocation, in addition to the features mentioned the gait is waddling; the lordosis very decided (Fig. 370); and the perineum and the distance between the thighs is widened. Apart from these symptoms there is sometimes pain in the joints and fatigue on walking, and in some cases inability to walk at all except with crutches.

The **diagnosis** is to be made from coxitis by the freedom of movement of the joint; and from coxa vara by earlier development, the absence of rickets, the absence of a right-angled inclination of the neck of the femur, and the presence of lordosis. Radiography will serve to establish the diagnosis of dislocation in doubtful cases. **Treatment** is surgical and of great importance. Various methods have been employed. The choice of those to be selected, whether with open incision or manipulative, has been a matter of much discussion, and is influenced to some extent by the age of the patient.

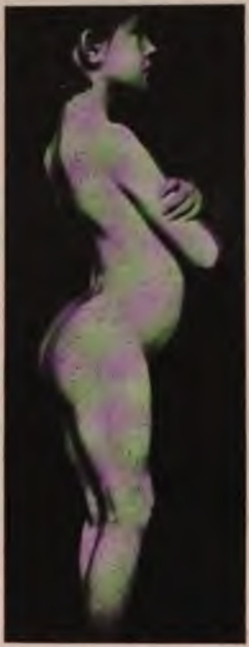

FIG. 370.—CONGENITAL DISLOCATION OF THE HIP. *Courtesy of Dr. H. R. Wharton.*

CURVATURE OF THE SPINE

Etiology and Symptoms.—Spinal curvature is of various sorts, usually not congenital in origin, and due to many causes. **Lordosis** is an exaggeration of the normal anterior curve in the lumbar region. It is observed in congenital dislocation of the hip (see Fig. 370), coxitis, paralysis of the muscles of the back, and sometimes in rickets and spinal caries. **Kyphosis** is an abnormal posterior curvature oftenest somewhere in the dorsolumbar region. Occurring in both lumbar and dorsal regions, but most marked in the former, it is especially well seen in rickets during the earlier stages (Vol. I, p. 594, Fig. 204). At a later period of life kyphosis, chiefly in the dorsal region, is very common in stooping, round-shouldered children. An angular kyphosis results from spondylitis. It may affect any portion of the spinal column, and is at first of but very limited extent. It may, howerer, become very marked, especially in the dorsal region, and produce a disfiguring hump-backed deformity. (See Tuberculous Spondylitis, p. 442, Fig. 386.)

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[1] Surgery of Childhood, 1910, 721.

Scoliosis, or lateral curvature (Fig. 371), is of very common occurrence, it being estimated that from 25 per cent. to 50 per cent. of all school-children exhibit some degree of the deformity (Bradford and Lovett).[1] Rarely it is congenital, and is then due to malformation of the ribs or vertebræ. Early cases are oftenest seen in rachitic children who have been carried by the nurse in an improper manner, perhaps always upon the one arm. Other causes are poliomyelitis; faulty position in standing, sitting or lying, as a result of habit or of certain occupations or even of sports; unequal length of the legs; contraction of the chest following empyema, etc. The curvature may be situated in any portion of the spine, and may be single and to either side; but, as a rule, there are other compensating curves present, and always there is also a rotation of the bodies of the vertebræ. The degree of scoliosis is usually so slight that it attracts little attention, and is only discovered when an examination of the bare back is made. It may, however, be so decided that great deformity results. With the curvature of the spine there are necessary alterations in the position of the pelvis, the hip on the side of the convexity being more prominent and the shoulder higher.

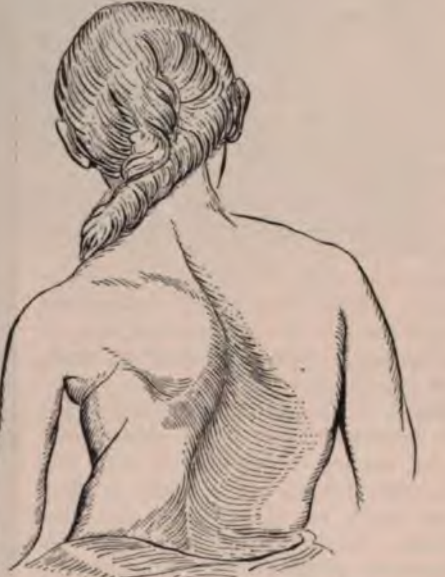

FIG. 371.—WELL-MARKED SCOLIOSIS.
(*Griffith, The Care of the Baby*, 1915, *6th Ed., p.* 304.)

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[1] Orthop. Surgery, 1915, 212.

Treatment.—All of the forms of curvature require early recognition and prompt and energetic treatment, since the ill effects are often far reaching. In cases where curvature is likely to occur, this lesion should be anticipated by the employment of prophylactic measures. For cases with curvature already present, the causes must be found and removed when possible. In many instances gymnastic exercises, massage, and the application of suitable apparatus are indicated. This subject is of such importance that reference for further details must be made to treatises upon orthopedic surgery.

The various spinal curvatures described, if well marked, are attended by corresponding bony deformities in other regions than the spine. There are deformities, too, produced in other ways which require mention here:

Congenital Elevation of the Scapula (*Sprengel's Deformity*).[1] (Fig. 372).—This is an unusual condition in which the scapula, oftenest on one side only, is displaced upward and rotated with the lower angle too near the spinal column. Sometimes a bridge of bone unites the latter to the scapula. The arm of the affected side cannot be raised above a right angle with the body. As a result of the malformation the head is inclined to this side and scoliosis is present. Other deformities may be combined with this. Massage and exercises may do good, and in other cases operative interference is required.

Winged Scapulæ (*Scapulæ alatæ*).—This may result from displacement of the latissimus dorsi; lateral curvature; muscular debility or wasting; or paralysis of the serratus magnus. The angle of the scapula is tilted outward causing the characteristic winged appearance. A somewhat similar effect is produced in pseudohypertrophic muscular dystrophy by the hypertrophy of the infraspinati and the abnormal mobility of the scapulæ.

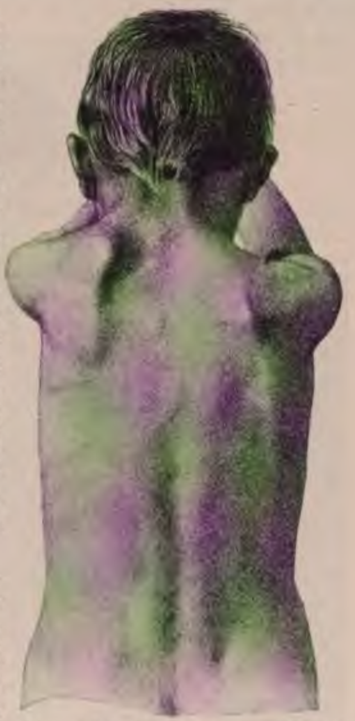

FIG. 372.—CONGENITAL ELEVATION OF THE SCAPULA.

Seen in the left shoulder. (*Sprengel, Arch. f. klin. Chir.,* 1891, *XLII,* 545.)

Pigeon-breast (*Pectus carinatum; Chicken-breast; Keel-shaped Chest*).—In this condition there is exhibited a prominence of the sternum and cartilages, with a depression of the sides of the thorax. It is an important symptom in rickets, but also results from prolonged interference with respiration, as from the presence of adenoids. Well-marked kyphosis from dorsal caries will likewise produce it. Treatment in all but the last mentioned is satisfactory, and consists in the practising of deep inspiration and of forced expiration, and in such gymnastic exercises as swinging from a horizontal bar, hanging on rings, and the like, which tend to pull out the sides of the chest and expand the lungs. Systematic pressure at intervals upon the sternum is also recommended.

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[1] Arch. f. klin. Chir., 1891, XLII, 545.

Funnel-chest.—This is a depression of a funnel-like form in the sternum. It may either be of considerable extent along the course of the sternum (*depressed sternum*) (see Vol. I, p. 689, Fig. 238), or much smaller and more abrupt, and be then truly funnel-form (Fig. 373). In some cases it appears to depend upon rickets or obstruction to respiration; and in others to be rather a congenital anomaly. Treatment for rickets or nasal obstruction, and the employment of suitable gymnastic exercises, as in pigeon-breast, and of forcible expiration against resistance may be of avail. The treatment is best conducted by an orthopedic surgeon.

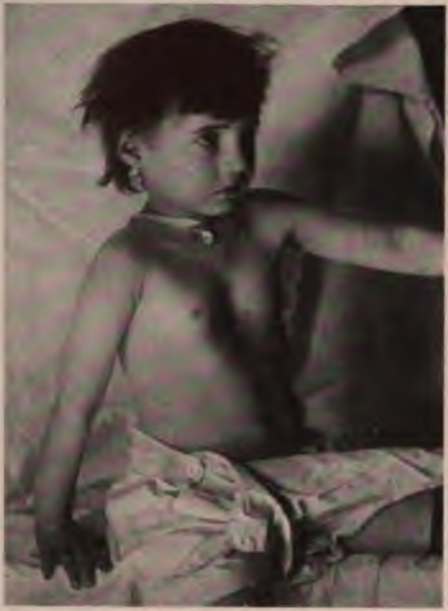

Fig. 373.—Funnel-chest.
From a patient in the Children's Hospital of Philadelphia.

CLUB-FOOT

(Talipes)

Etiology.—Of the various deformities of the extremities this is one of the most frequent. It may be congenital, occurring once in about 1000 births (Willard).[1] There is often a distinct inheritance observed, and other malformations are frequently present. The disease is much more common in boys, and it is oftener than not bilateral. The congenital cases are probably produced by abnormal intra-uterine pressure, or by other processes within the uterus. The acquired cases are about 3 times as numerous as the congenital; and are the result of trauma or of disease in the neighborhood of the ankle; of cerebral paralysis; and especially of poliomyelitis.

Symptoms.—The four chief forms are *talipes varus*, with the foot adducted and inverted, and the child walking on the outer border; *talipes valgus*, the reverse of this, walking taking place on the inner border; *talipes equinus*, the heel being turned up and the child walking on tip-toe; *talipes calcaneus*, the opposite condition, walking being on the heel with dorsal flexion of the foot. There may further be combinations of some of these. The most frequent congenital variety is equino-varus; while equino-varus and equinus are most common in the acquired cases.

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[1] Surgery of Childhood, 1910, 664.

When the child with talipes begins to walk, the deformity of the bones grows progressively greater, and the degree of the contraction of some muscles and of the feebleness of others increases; and, if the condition is allowed to continue too long untreated, the talipes finally reaches a state which is beyond cure. Consequently, the earlier treatment is commenced the better will the prognosis be. This treatment is very varied, and may be either manipulative, mechanical or operative, depending upon the case. It comes entirely within the province of the orthopedic surgeon.

FLAT-FOOT

(Pes valgus)

The very common deformity bearing this name demands recognition by the pediatrist. It may be congenital; develop early as the result of rickets; or be acquired by learning to walk too soon. Later it may be produced by improper foot-wear, especially tight shoes and high heels; general muscular weakness; too much continuous standing; or poliomyelitis. It is also often associated with knock-knee. In this malformation the arch, generally of both feet, is lowered or even entirely destroyed as a result of the weakness of the muscles and ligaments, and the inner aspect of the ankle is too prominent (Fig. 374). The child walks heavily and with the toes turned out too much; and seems to have weak ankles. The shoes wear out chiefly upon the inner side of the sole and over the internal malleolus. Fatigue occurs readily and there is often pain in the feet and sometimes in the back as well; and in severe cases the pain may continue even when the patient is off of his feet.

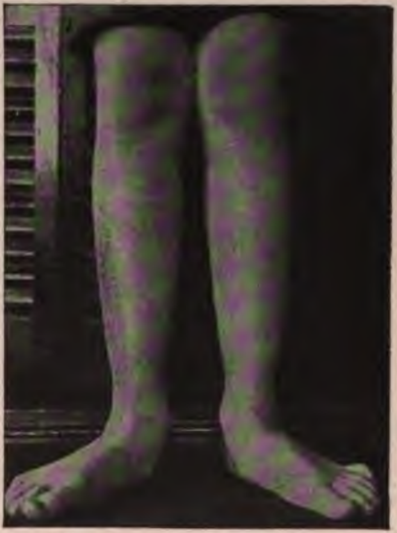

FIG. 374.—PRONATED FEET (FLAT-FOOT). (*Willard, Surgery of Childhood*, 697, *Fig.* 640.)

The **diagnosis** is rendered probable by the presence of the symptoms described, but careful examination of the whole of the lower extremity is required to confirm it, and it is to be borne in mind that other deformities of the feet, especially an unduly high arch, are likewise capable of producing pain (Lovett).[1] Examination of the foot while the child stands upon a table will reveal the depression of the arch; except in young children, where the fatty tissue normally present may be misleading. Yet even in these there is an abnormal showing of the pink sole upon the outer edge of the foot. Search should always be made for knock-knee.

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[1] Pediatrics, 1916, XXVIII, 16.

Treatment is satisfactory except in the severer, long-continued cases, and even here improvement can be obtained. Prophylaxis is of great importance. Infants should be taught to creep, and early walking discouraged; and this is particularly true if the child is rachitic or abnormally heavy. The shoes should be easy and with broad soles. Later the use of sandals or the going bare-foot is of advantage, since it develops the muscles of the foot. Should evidence of flattening appear, there should be made a thickening of the sole of the shoe upon the inner side, in order to throw the weight of the body on the outer border of the foot, and to keep the toes from turning outward when walking. If this does not answer, a slight curved elevation of leather may be inserted as an in-sole to support the weakened arch. The muscles of the foot should be strengthened by massage, and forcible frequent adduction of the foot by passive movements practised, and such exercises prescribed as walking without shoes on tip-toes with the toes turned inward. For more advanced cases a sole-plate may be necessary; but this should be prescribed accurately by an orthopedic surgeon.

PIGEON-TOES

The normal child while first walking tends to keep the feet parallel or even to turn the toes in (Fig. 375). Sometimes this persists as a habit for an unusual time, and then causes the mother anxiety. It is not, however, to be discouraged in infancy, as it will correct itself in due course; and too early turning out of the toes is liable to favor the production of flat-foot and knock-knee. This statement does not oppose the instituting of a thorough search for possible causes which may need correction. The condition may depend upon knock-knee, a certain degree of club-foot, or an inward rotation of the hips; and should any such cause be suspected, the advice of an orthopedist is necessary.

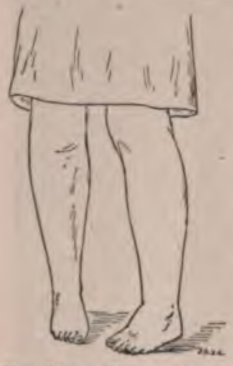

FIG. 375.—PIGEON-TOES.

(*Griffith, The Care of the Baby*, 1915, *6th Ed.*, *p.* 301.)

KNOCK-KNEE

(Genu valgum)

This is a common deformity, usually resulting from rickets, general debility, poliomyelitis, or from a child being carried in a faulty manner. These produce relaxation of the muscles and ligaments, with consecutive deformity of the adjacent extremities of the femur and tibia. The internal condyles are always enlarged. Either one or both legs may be affected, usually one more than the other.

Symptoms.—When both legs are involved the child stands with the knees close together and the ankles apart (Fig. 376). In early cases the feet in walking are turned in, but in those more pronounced there is always some degree of talipes valgus, the toes being turned out unduly and the patient flat-footed. The gait is somewhat waddling, or in unilateral knock-knee there is a limp. The child suffers readily from fatigue and sometimes from pain. It is to be noted, however, that a large proportion of children are temporarily knock-kneed during the process of learning to walk. In contra-distinction to the pathological knock-knee, such children consistently turn their toes in, and this is in no way to be interfered with. The deformity at the knee will soon disappear.

Treatment is of great importance. In the way of prophylaxis, the rickets which is so frequently present must receive appropriate management, and standing and walking be delayed as much as possible. Should knock-knee begin to appear, the inner border of the sole of the shoe should be thickened, as in cases of flat-foot, in order to throw the knees out and turn the toes in; and if necessary the arch of the foot supported as well. Garters should not be worn suspending the stockings from the outer side of the legs. If the patient is still quite young, and the bones soft, much can be accomplished by forcible bending of the knees outward while the leg is extended at the knee-joint. This should be done several times a day, using as much pressure as the child can tolerate. Walking barefoot and on the tips of the toes is of benefit, as in cases of flat-foot. The child should be taught to stand with the heels close together, and walking with the toes turned in should be favored. In older children bicycle riding and horse-back riding astride is of service. A great many cases will recover under treatment of this sort, if it is commenced early. Should, however, such measures not avail, mechanical treatment must be applied by an orthopedic surgeon.

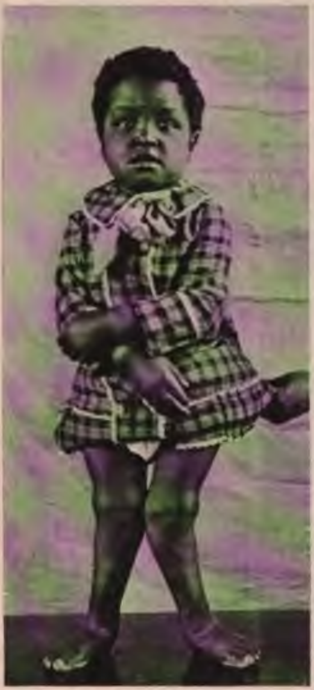

FIG. 376.—KNOCK-KNEE.
Courtesy of Dr. H. R. Wharton.

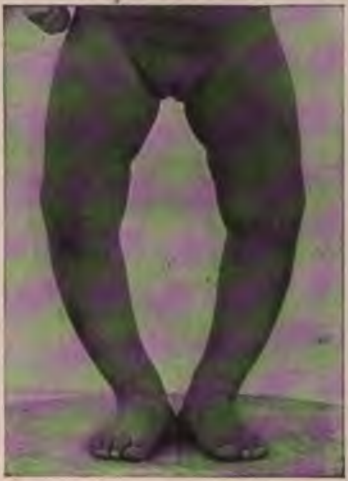

FIG. 377.—OUT-KNEE AND BOW-LEGS.
Resulting from too early walking while rickety bones were soft. (*Willard, Surgery of Childhood,* 301, *Fig.* 252.)

BOW-LEGS

(Genu varum)

This is the reverse of knock-knee and is of very common occurrence. All infants in the earliest months of life exhibit a certain degree of normal bowing, which, however, soon disappears, unless the condition is very marked. (See Vol. I, p. 19.) The usual cause of bow-legs is rickets, especially if children with this disease are permitted to stand or walk early. Even without rickets, bow-legs may result from the wearing of thick diapers.

Symptoms.—Under the influence of the weight of the body, the soft bones of the legs and thighs of both sides become curved outward. When the child is standing the knees are widely separated, and the feet tend to assume the position of talipes varus (Fig. 377). Sometimes one leg is bowed and the other exhibits knock-knee—a condition resulting from the pressure of the arm of the nurse carrying a rachitic child always upon her one side. Sometimes, too, the bones of the legs may be bowed, yet without the knees being bent outwardly. In severe rachitic cases there may be both bowing and anterior bending, produced by the habit of sitting with the legs crossed or by too early bearing of weight upon them. A careful examination should be made of the child undressed, and both while standing and when lying upon the back, in order to determine just what bones are most bent.

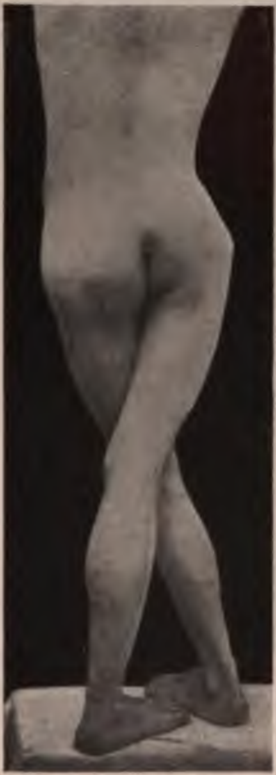

Fig. 378.—Coxa Vara. Showing "scissor-leg" deformity. (*Young, Orthopedic Surgery*, 1906, 393, *after Morton*.)

The **prognosis** is good, except in severe advanced cases. A great many children recover spontaneously if the deformity is slight. **Treatment** should be prophylactic, especially the prevention of too early standing in the case of rachitic children, and the employment of measures to hinder the development of this disease. In children with bow-legs whose bones are still soft, firm pressure should be made against the curvature several times a day. Massage should also be given. If these remedies do not avail promptly, it may be necessary to apply mechanical apparatus, or to resort to some operative procedure. Treatment of this sort is naturally in the hands of the surgeon.

COXA VARA

In this disorder the neck of the femur is at a right angle or even an acute angle with the shaft of the bone. The condition may be either bilateral or unilateral; is rarely congenital; often is the result of rachitic softening or of epiphyseal separation, with later union at an improper angle; still oftener (*coxa vara statica*) develops in later childhood or adolescence, and is dependent upon a yielding of the neck of the femur from various causes, such as poliomyelitis, disease of the epiphysis, or trauma with fracture (*coxa vara traumatica*).

Symptoms.—In the unilateral cases these consist in pain in the affected hip, often extending to the knee; a limping gait; shortening of the limb and of the adductors of the thigh; limited power of abduction; inward rotation of the hip; upward tilting of the pelvis on the affected side, and upward displacement of the trochanter above Nélaton's line. When the disease is bilateral, there is a waddling gait as in congenital dislocation of the hip. The knees frequently pass each other with difficulty in walking (*scissors-gait*) (Fig. 378). The diagnosis is to be made from congenital dislocation of the hip by the later period of development; the firmer gait; the decided knock-knee; the absence of lordosis; and the presence of the head of the femur in the normal position. Hip-joint disease exhibits a general fixation of the hip, while in coxa vara there is limitation only in abduction. A radiograph should always be made. The treatment is entirely surgical, except that if the diagnosis is made early, walking should be entirely prevented.

HYPERTROPHIC PULMONARY OSTEO-ARTHROPATHY

(Clubbing of the Fingers)

This disorder, so named by Marie,[1] is the result of prolonged suppurative disorders, such, especially, as empyema, bronchiectasis, and chronic phthisis; but it may occur also in congenital cardiac disease, and is sometimes seen with syphilis. The method of production is not clearly understood. It has been supposed to be dependent upon toxins derived from the pus, but this fails to explain its development in congenital affections of the heart. The lesions consist in a deposit of subperiosteal bone, the result of an ossifying periostitis. There is also effusion into the joint, erosion of the cartilages, and hypertrophy of the soft tissues. In the well-developed cases there is very decided enlargement of the ends of the long bones and of the hands and feet, with pain and swelling in the joints; but in the milder instances, which constitute the majority especially in children, the disease is manifested chiefly by marked clubbing and enlarging of the ends of the fingers, the nails being broad and curved in form transversely and longitudinally (Fig. 379). (See also p. 126, Fig. 307.) A distinction has been made between the condition limited to clubbing of the fingers and toes (Hippocratic fingers), in which there is said to be no changes in the bones, and the more severe condition in which the terminations of the long bones are involved. Inasmuch as the causative diseases are for the most part the same, and clubbing of the fingers always accompanies the other lesions, it is questionable whether any sharp

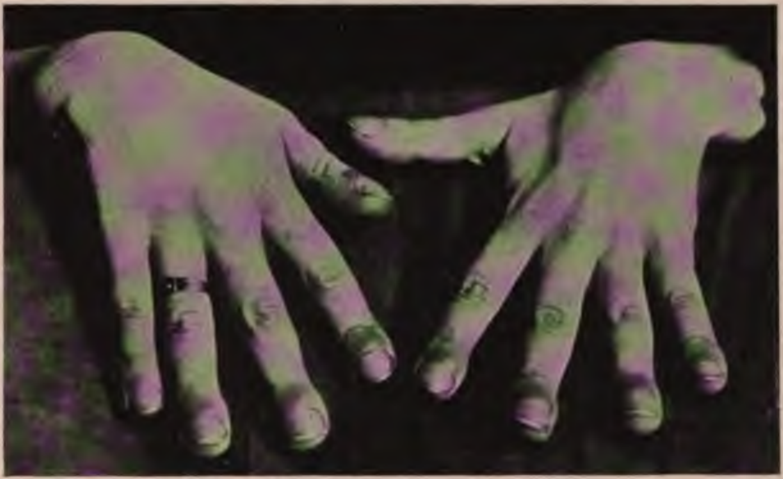

FIG. 379.—PULMONARY OSTEO-ARTHROPATHY.

From a patient in the Children's Hospital of Philadelphia, with abscess of the lung, the result of the entrance of a water-melon-seed into a bronchus.

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[1] Rev. de méd., 1890, X, 1.

distinction can be drawn. Moreover, as pointed out by Locke,[1] a considerable percentage of cases of apparently simple clubbing exhibit also involvement of the long bones, as proven by *x*-ray examination.

CRANIOTABES

By this term, first applied by Elsässer,[2] is designated a condition of thinning of the bones of the cranium in spots. Rickets is one of the common causes; yet it would appear probable that hereditary syphilis is a more frequent factor. Carpenter[3] found in 238 cases that 87 per cent. were syphilitic and that 61.3 per cent. occurred in the first 4 months of life, at a time when rickets has not, as a rule, become well developed. It is possible, too, that in some cases errors in diet give rise to the spots before or without the development of rickets, and Leroux and Labbé,[4] while confirming the preponderating influence of syphilis, believe that other causes likewise may be operative. The production of the lesion is by an absorption of bone, beginning in the inner table and perhaps extending so completely through the thickness of the skull that only membrane remains, which yields readily to pressure by the fingers. The spots are situated oftenest in the parietal bones; less often in the occipital, either with no relation to the sutures, or frequently toward the edge of the bone and especially near the parieto-occipital suture. The condition disappears as the children grow older and the health improves.

ACHONDROPLASIA

(Chondrodystrophia fetalis)

This is the title applied by Parrot[5] to a condition of dwarfism, not common, yet observed for centuries. There is reason to believe from sculpture that it had been recognized in ancient Egypt; and many of the dwarfs pictured in art were of an achondroplasic type. A series of interesting older illustrations is given by Regnault.[6] The name chondrodystrophia fœtalis was applied to it by Kaufmann.[7]

Etiology.—The cause is entirely unknown. Tuberculosis and syphilis have no connection whatever with it. The same is true of rickets, and the distinction between the two disorders has been clearly shown by many. Undoubtedly very many of the cases which have been described as fetal rickets were, in fact, instances of achondroplasia. (See Rickets, Vol. I, p. 598.) It has been supposed by some observers that the disease depended upon a disordered internal secretion of the thymus or thyroid gland, while others placed it among diseases of the nervous system. Heredity has been shown in a number of instances to play a part through the father, a mother with achondroplasia being seldom able to give birth to living children.

Pathological Anatomy.—The process begins in early fetal life, and consists in a failure of proper development of the ossifying process in the cartilaginous tissue, especially that of the long bones. As a result there occurs finally a premature ossification of the cartilage, and the long bones are shorter than normal, but not thinner, since the production of bone taking place beneath the periosteum is not interfered with. The cartilage-cells of the proliferating zone are diminished in number. There is a disturbance of the columnar arrangement of the cells, the cartilage is vascular, and between the epiphysis and the diaphysis there is a fibrous extension from the periosteum which aids in checking the growth of the bones in length. In some cases there is hyperplasia of the cartilage-cells of the epiphysis resulting in enlargement of this, and in still others the epiphysis is softened; but in the majority it is normal in size, and the increase in size of the ends of the bones is due to a cup-shaped enlargement of the diaphysis around the epiphysis (Fig. 380). The cartilaginous bones of the base of the skull are involved in the process, and in consequence there is an early ossification of these and a failure to reach a fully developed size. This produces a distortion of the position of the remaining membrane-bones of the skull. These, as well as the vertebræ and the clavicles, are uninvolved in the process.

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[1] Arch. Int. Med., 1915, XV, 659

[2] Der weiche Hinterkopf, 1843.

[3] Rep. Soc. Study of Dis. of Child., 1903, III, 214.

[4] Ann. de méd. et de chir. inf., 1912, XVI, 481.

[5] Bull. soc. d'anthrop. de Paris, 1878. La syphilis héréd. et la rachitis, 1886, 280.

[6] Arch. de méd., 1902, VII, 232.

[7] "Untersuchungen ü. d. sogen. Rachitis fœtalis," 1892. Ref., Centralbl. f. Gynæk., 1892, XVI, 358.

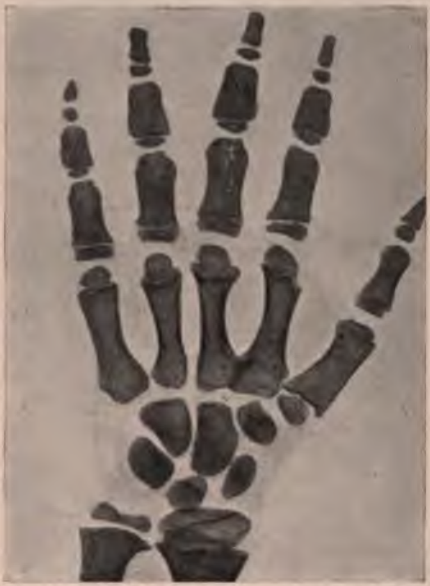

FIG. 380.—RADIOGRAPH OF THE HAND IN ACHONDROPLASIA (POSTERO-ANTERIOR VIEW).

Exhibits the expansion of the diaphysis of each of the long bones, and the stunting and increased breadth of the metacarpals and phalanges. The divergence of the middle and ring-fingers is seen to be due to excessive expansion of the heads of their proximal phalanges. (*Balme and Reid, Practitioner,* 1904, *LXXIII,* 780, *Fig.* 4.)

Symptoms.—The symptoms are visible at birth. The chief characteristic is the stunted condition of the extremities, while the trunk is normal in length (Fig. 381). The shortness in the thighs and arms is greater than in the legs and forearms. The hands may not reach below the hips, and are short and broad, and the fingers are thick, short and spreading and of nearly equal length, with a separation between the second and third fingers, producing what has been called the "trident hand" (Fig. 382). The long bones of the body are not only short and thick, but often sharply curved at the epiphyseal junctions. There is enlargement at these junctions, and the ribs are beaded. The skin of the limbs shows folds and deep furrows, as though it were too abundant (Fig. 383). This is especially marked on the extensor surface of the lower extremities. These furrows disappear as the patient grows older. There is generally a large amount of subcutaneous fat. The abdomen is prominent; and the head appears large and brachycephalic, and with prominent forehead, flattening of the bridge of the nose, and a projecting forward of the lower jaw. The lips are thick, and the fontanelle is late in closing, this perhaps not taking place until the child is several

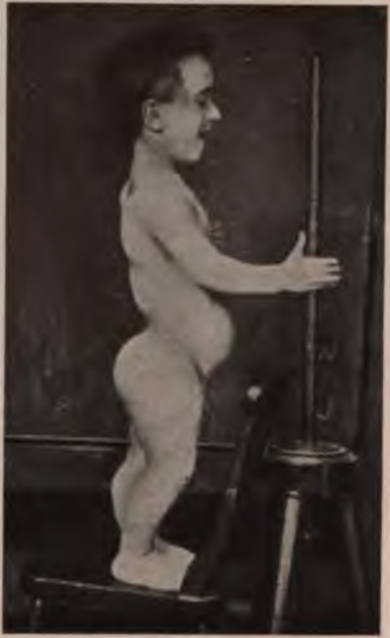

FIG. 381.—ACHONDROPLASIA.

Boy of 12 years. Condition noticed at birth. Height 38¾ inches. Intelligence normal. Illustration shows the depression of the bridge of the nose, the expanded cranial vault, and the marked lordosis. (*Balme and Reid, Practitioner*, 1904, *LXXIII*, 790.)

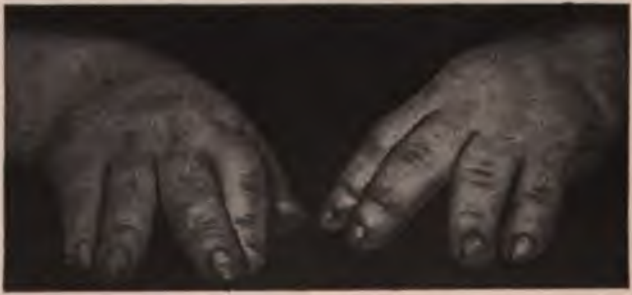

FIG. 382.—THE "TRIDENT HAND" IN ACHONDROPLASIA.

(*Fussell, McCombs, deSchweinitz and Pancoast, Journ. Amer. Med. Assoc.*, 1909, *LIII*, 1614.)

years old. Complicating malformations of certain sorts are not infrequent; among them hydrocephalus, polydactylism and inguinal hernia.

Course and Prognosis.—The majority of achondroplasics are stillborn or die very soon after birth. The remainder are liable to be delicate during the 1st year, but after this the duration of life is not affected by the disease. Dentition occurs at the usual time, but walking is often learned very late, perhaps not until the age of 3 or 4 years. Growth of the body takes place slowly and never normally, and the final height is seldom over 4½ feet. As infancy and childhood are passed, the subjects often become very muscular. The gait is frequently waddling, the legs bowed, and there is lordosis; and these, with the prominent abdomen and nates, produce a very characteristic appearance. The pelvis is contracted; dentition is unaffected; the sexual functions are normal. The mental faculties are somewhat slow in developing; speaking, reading and other intellectual acts being acquired rather later than usual, but the intellectual power is finally normal, or somewhat below this.

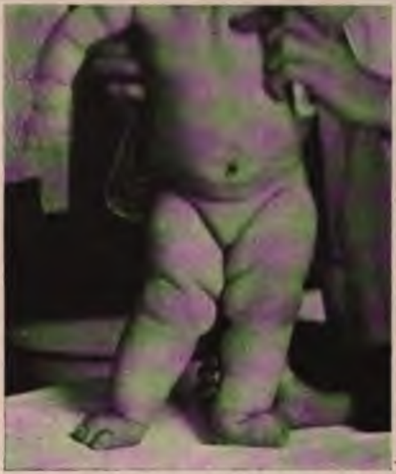

FIG. 383.—ACHONDROPLASIA, SHOWING FOLDS OF THE SKIN ON THE LOWER EXTREMITIES. From a patient in the Children's Hospital of Philadelphia.

Diagnosis.—In cases past the period of infancy or early childhood this is usually easy. At first the disease strongly suggests *cretinism.* In this, however, there is at the beginning no lack of proportion between the limbs and the trunk; and no prominence of the buttocks or characteristic alteration of the shape of the face and head, while fat-pads are present in the region of the clavicles. The depression of the bridge of the nose in cretinism develops later. Achondroplasics have normal thyroid glands and exhibit a mental power which is normal or, at the worst, far in excess of that of cretins. *Rickets* has often been confounded with achondroplasia, and repeatedly cases of achondroplasia have been described as "fetal rickets." In fact the disease was earlier known only by this name. There is in reality little in common between them. In rickets there is delayed dentition; no early deformity of the face and head, and no disproportion between the extremities and the trunk; while there exists a characteristic alteration of the shape of the thorax. In both conditions there is enlargement of the ends of the bones. It is possible for an achondroplasic to be rachitic as well. *Infantilism* or dwarfing from other cause does not exhibit the disproportions seen in achondroplasia. The forms of *osteogenesis imperfecta* as presently to be described, constitute an entirely different affection. The lesions in this disease are not in the cartilage at the epiphyseal junction, but in the shafts of the long bones or in the membrane-bones. As a result, there is a tendency to multiple fractures; to a membrane-like condition of the bones of the cranium; or to softening. The shortness of the limbs often seen is dependent upon the fracturing with union in malposition.

The **treatment** of achondroplasia is unavailing. No method hitherto tried has been of any benefit.

IDIOPATHIC OSTEOPSATHYROSIS

(Fragilitas Ossium)

Fragility of the bones in early life may occur secondarily to several other conditions, such as scurvy, rickets, and inherited syphilis. These are not under consideration here, but only the tendency to the occurrence of multiple fractures without sufficient discoverable cause. The cases of multiple intrauterine fractures combined with imperfect development of the membrane-bone are considered under Osteogenesis Imperfecta (p. 433). The two conditions are, however, clearly allied. Osteopsathyrosis is only occasionally seen, yet there are a large number of cases on record. Writing in 1897[1] I collected from medical literature 67 cases, including 1 very characteristic example under my own observation; and I have since seen several instances of it. Some of the collected cases, however, should more properly be placed in the category of osteogenesis imperfecta. Ostheimer[2] has added to this list 81 cases, excluding those placed by him under osteo-genesis imperfecta.

Etiology and Pathological Anatomy.—The cause is entirely unknown, except that heredity and familial influence play a part in a considerable number of cases. Brothers and sisters suffered fron the disease in 29.6 per cent. of Ostheimer's series. The condition was reported by Spurway[3] in 4 generations. The pathological lesions in the typical cases appear to vary, and are not clearly understood. No changes whatever in the chemical or anatomical condition of the bones have been detected in some instances, unless it be a slenderness of the shafts and thinness of the cortex. By one observer the marrow-cavity was found nearly obliterated; by another the organic material of the bone in excess; by still another the substance of the bone unusually porous with large marrow-spaces. A deficiency of osteoblasts has been claimed to exist by some; a lack of calcium by others; an incomplete formation of bony tissue by still others. Some of the descriptions are of cases which must be regarded as mixed ones, and to a large degree they are contradictory; and it would seem certain that we do not well understand the pathological anatomy of this disease. There are, of course, thickenings and deformities, the result of callus-formation and angular union following fracture.

Symptoms.—These consist solely in the extreme fragility. Fracture may occur previous to or during normal labor, and after birth entirely inconsiderable movements occasion it, such as the mere lifting of the child from the bed. It may be necessary to keep the infant recumbent and to carry it upon a stretcher. The fractures are oftenest situated in the long bones, ribs and clavicles, may be complete or of the green-stick variety, occasion less pain than usual with this lesion, and unite readily with abundant callus, but with malposition, shortenings and deformities of all sorts. The symptoms as described represent the condition in its most typical state. In the case previously reported by me a child of 2 years and 10 months had the history of having sustained 17 or 18 fractures. By no means all cases, however, exhibit so marked a degree of fragility. It seems probable that fragilitas may be *combined* with the osteogenesis imperfecta to be described, while other cases possess a certain degree of mollitics ossium, as a result of which the bones are deformed not only by fracturing but by bending through softening. An interesting symptom sometimes observed is a dark-blueness of the scleræ. I have seen this in some instances under my care. The association of this with fragility was first mentioned by Eddowes,[1] and the subject has been given attention by a number of writers (Hofmann;[2] Herrman;[3] Bronson[4]).

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[1] Amer. Jour. Med. Sci., 1897, April.
[2] Jour. Amer. Med. Assoc., 1914, LXIII, 1996.
[3] Brit. Med. Journ., 1896, II, 844.

The **prognosis** of the disease is unfavorable, so far as the disappearance of the brittleness is concerned, although this may diminish. The condition is not dangerous to life, except in the severe cases developing early, many of which die from the ready attendance of complications. Others may remain severely crippled and perhaps bedridden.

The **diagnosis** is readily made. *Rickets* produces bending of the bones, without complete multiple fractures. There are also beading of the ribs, enlargement of the epiphyses, and other characteristic manifestations. Inherited *syphilis* occasionally brings about a separation of several epiphyses which may at first suggest fracture. The situation of this lesion is characteristic. *Achondroplasia* has shortening, but without fracture. *Osteogenesis* imperfecta exhibits distinct evidences of imperfect bone-formation, and incomplete development of the membrane-bones of the vault of the cranium.

The only **treatment** possible is the employment of extreme care in the handling of the affected infant. Fractures which occur should be managed as is usual for such traumata. The general health is to be maintained, and gentle massage is of service in this direction.

OSTEOGENESIS IMPERFECTA

(Periosteal Aplasia)

In a broad sense achondroplasia, osteopsathyrosis, and certain other affections could properly be included under this heading on the ground that they possess in common a defective bone-formation. Inasmuch, however, as some of them exhibit such very distinct clinical and pathological features, it seems best for convenience of description and to avoid confusion to limit the term osteogenesis imperfecta to certain cases with defective osseous development of intra-uterine origin best marked in the membrane-bones, although observed also in other regions. Ostheimer[5] has collected 44 cases from medical literature, in addition to a number included by me[6] in a publication upon osteopsathyrosis but which should probably be placed here.

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[1] Brit. Med. Journ., 1900, II, 222.

[2] Arch. f. klin. Chir., 1915, CVII, 279. Ref., Brit. Jour. Child. Dis., 1916, XIII, 317.

[3] Amer. Jour. Dis. Child., 1915, IX, 205.

[4] Edinb. Med. Journ., 1917, I, 240.

[5] Journ. Amer. Med. Assoc., 1914, LXIII, 1996.

[6] Amer. Jour. Med. Sci., 1897, April.

The **cause** is entirely unknown, but the disease at least has nothing in common with rickets or syphilis. The disorder is encountered oftenest in fetuses or still-born infants, but may be present in those who survive birth. The basis of the **lesions** is a defective development of bony tissue both endochondral and periosteal, but especially the latter; and the cartilage itself is not materially altered in its character. Few normal osteoblasts are found. As a result of this defective osseous development the vault of the cranium may suggest on examination a soft-shelled egg, or a limited deposition of osseous material may produce a crackling sensation when the head is handled. The long bones, ribs and clavicles may be soft and fragile, bending readily in any direction, or breaking under slight pressure, and they exhibit many deformities. Irregular thickenings may be felt in these bones, suggesting union after fracture with abundant callus. This is probably the real explanation in some cases; while in others the nodules depend merely upon irregularities in ossification. In a case dying 24 hours after birth reported by Chaussier[1] there were found 113 fractures. The deformities of the extremities resulting from the pathological process produce often a decided shortening of them; but in a limb which has escaped these changes the length is normal. The **prognosis** is entirely unfavorable. In a case seen by me in which the bony vault of the cranium was almost completely membranous, life continued until the age of 13½ months; but as a rule death takes place much earlier than this.

The **diagnosis** is readily made. The disease differs from osteopsathyrosis chiefly in the thinness of the vault of the skull combined with the deformities of the long bones which may or may not be associated with fracture. Many of the cases called "fetal rickets" are really instances of osteogenesis imperfecta. In achondroplasia the process is at the epiphyseal junctions, not in the shafts, and there are no fractures.

Treatment is of little avail. The administration of cod-liver oil, phosphorus and calcium seems indicated.

CLEIDOCRANIAL DYSOSTOSIS

The rare affection brought into prominence by Marie and Sainton[2] has been studied with care by Fitzwilliams[3] who analyzed 60 collected reports of cases in addition to 45 others previously studied by Villaret and Francoz.[4] The cause is entirely unknown, except that the affection is often distinctly hereditary or familial. Langmead[5] reported the occurrence of 18 cases in 4 generations. It is allied to osteogenesis imperfecta in that it consists in a defect in the formation of the membrane-bone. In the skull this is shown by delay in the ossification of the bones of the cranium, with the persistence to adult age of open anterior and posterior fontanelles. Large bosses develop on the frontal, parietal and occipital regions. The skull is brachycephalic and globular; the forehead protruding; the bones of the face small; the arch of the palate high and perhaps cleft; the dentition irregular. The deformities of the clavicles are of especial interest. These bones may be absent altogether, or entirely cartilaginous; or the sternal ends may be present and the remainder absent; or the distal portions may not be united to the adjacent parts. The malformation of the clavicles permits of the shoulders being drawn together in front to a remarkable degree (Fig. 384). Patients with these malformations are generally shorter than normal, but with intellectual powers and general health unaffected.

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[1] Bull. de la Faculté de méd. de Paris, 1814, III, 306. Ref., Harbitz, Beit. z. path. Anat. u. z. allg. Path., 1901, XXX, 630.

[2] Bull. et mem. soc. méd. des hôp. de Paris, 1897, 3, XIV, 706; 1898, XV, 436.

[3] Lancet, 1910, II, 1466.

[4] Nouvelle iconographie de la Saltpêtrière, 1905, XVIII, 302.

[5] Proc. Roy. Soc. Med., 1916, X, 1. Dis. Child., 1.

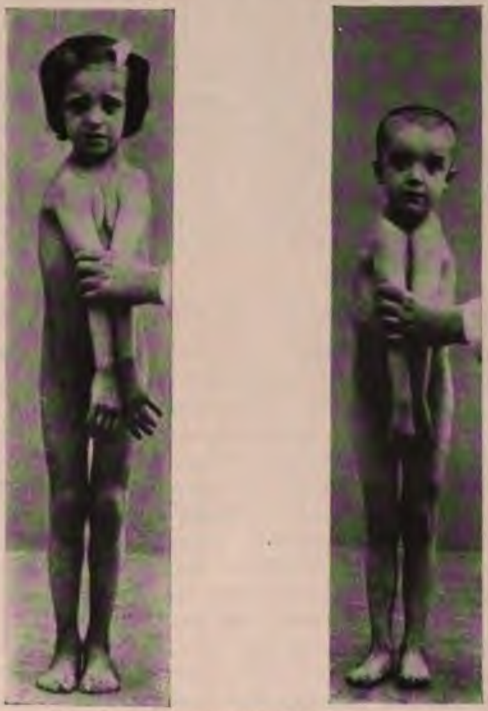

FIG. 384.—CLEIDO-CRANIAL DYSOSTOSIS.

Sister and brother aged 9 and 6 years respectively. Illustrates broadness of the head and extreme mobility of the shoulder-joints, the result of imperfect development of the clavicles. A younger sister and the mother exhibited the same symptoms to a lesser degree. (*Villaret and Francoz, Nouvelle Iconographie de la Saltpêtrière*, 1905, *XVIII*, 302.)

OSTEOMALACIA

(Mollities Ossium)

This is an acquired condition very rarely observed in children. It consists in a softening of the bones analogous to that seen in rickets. Although described in infants by Rehn[1] and others, that it ever occurs in them seems doubtful; and there would be in any event no way to distinguish its symptoms at this period from those of rickets. In older children it has been said to have followed attacks of the infectious diseases; and possibly the occurrence of rickets early in life may be productive of mollities later. Very possibly, too, some cases which have been called "late rickets" (Vol. I, p. 598) were, in fact, osteomalacia. The softening has also been attributed to the influence of the internal secretion of the ovaries or of the suprarenal glands. Mollities may occur, too, as a complication of osteopsathyrosis and of osteogenesis imperfecta.

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[1] Jahrb. f. Kinderh., 1897, XII, 100.

The **lesions** comprise a thickening of the periosteum, with softening of the bone beneath through the absorption of the calcium salts and the occurrence of an osteitis. The medullary cavity is enlarged. The bone can be cut easily with a knife. The early **symptoms** consist in the gradual development of pain in the bones, especially those of the legs, made worse by exercise or by pressure. Bending then develops and may become very great, and fractures may later take place. The course is often long and progressive, although periods of remission may occur. The prognosis is unfavorable, yet recovery may follow in exceptional instances. The disease is distinguished from rickets by the later period at which softening takes place, and from osteopsathyrosis by the absence of early fracture or evidence of actual brittleness. In the line of **treatment** bending and fractures are to be prevented as far as possible, and the general health maintained. Adrenaline has been of some value in adults, and phosphorus, calcium salts and cod-liver oil have also been recommended.

INFLAMMATION OF THE BONES AND JOINTS

Any systematic classification of these conditions is far from satisfactory, and the titles generally employed by writers are confusing; one condition often being called by many different names; or one title being applied to different forms of inflammation. So, too, it is impossible to make a sharp distinction between diseases of the shafts of the bones, the epiphyses, and the articulating surfaces, because an inflammation arising in any one part may readily extend to others. On the other hand, a purely pathological classification separates too widely inflammations which are symptomatically much the same. The following division is therefore intended only as a tentative one. The subject is so largely surgical that it can be considered here but briefly, and with omission of many topics. That reference is made to any of them is due to the fact that in their early stages they come under the observation of general practitioners rather than of surgeons; and some knowledge is necessary of their symptomatology and diagnosis as occurring in children.

ACUTE INFECTIOUS OSTEOMYELITIS

(Acute Epiphysitis; Acute Septic Diaphysitis; Acute Periostitis)

Etiology and Pathological Anatomy.—The disease consists of an acute pyemic infection of the bones. It occurs oftenest in males in later childhood, although it is not uncommon somewhat later or earlier than this, and may be seen even in infancy. It develops only during the period in which the bone is still quite vascular and is growing. Under the influence of trauma; of some neighboring local source of infection; or of some debilitating or infectious disease, septic germs enter from the circulation, perhaps into a small clot which has formed in the bony tissue. The species oftenest present is the staphylococcus aureus; but the staphylococcus albus, streptococcus and pneumococcus are also met with. The disease begins in the marrow of the shaft of the bone near the epiphysis, in the portion where active growth is taking place, and an abscess forms. If life continues, the infection spreads by way of the Haversian canals to the periosteum (*acute periostitis*), allowing pus to collect beneath it, and stripping it from the shaft to a considerable extent; or the pus may extend throughout the whole of the marrow of the shaft (*osteomyelitis*). The epiphysis may be partially detached, and in other cases the pus may penetrate into it (*acute epiphysitis*) and thence into the joint (*septic arthritis*). Exceptionally the inflammation starts beneath the periosteum. The rapidity of the process in severe cases of osteomyelitis is often very great, and in only a few hours from the onset the periosteum may have been lifted from the bone.

As a result of the septic inflammation more or less of the bone becomes necrosed, and this portion, in the form of a sequestrum, is finally separated from the remaining healthy bone in the more chronic cases, by the reparative development of granulation tissue, while the periosteum produces new bone about it. Meanwhile the pus has penetrated in places through the soft tissues, producing sinuses. In favorable cases the sequestra are finally softened and discharged, or can be removed by operation, and recovery takes place; although the medullary canal of the shaft may be obliterated; and the destruction of the cartilage of the epiphysis may interfere with further growth of the limb.

The favorite situations of the lesions are the terminal portions of the long bones, especially about the knee; less often about the shoulders, ankles and elbows, and in the ribs. Only exceptionally are the short bones of the body attacked. The lesions of pericarditis, pleuritis, or multiple abscesses may be present in severe cases.

Symptoms.—The onset is sudden, perhaps with a convulsion or chilliness followed by high fever, delirium, and severe localized pain and tenderness, with slight redness and swelling. In the rapidly fatal cases the local manifestations may not pass beyond these, and the child may promptly develop evidences of severe sepsis, with pallor; diarrhea; sweating; rapid pulse; continued chilliness and fever; leucocytosis; and, finally, coma; a pericarditis often being an attendant complication. If the virulence of the case is not so great and the progress slower, the pain, swelling and redness extend over the whole section of the limb; and if the joint becomes involved, the symptoms of arthritis are added. This extension is especially liable to occur at the hip. Sometimes, too, the infectious material produces a metastatic multiple osteomyelitis if carried to other bones. In the more chronic cases, multiple sinuses form leading to the necrosed bone; the acute manifestations subside, and the patient may exhibit the symptoms due to prolonged suppuration, among them anemia, progressive loss of strength and weight, and possibly the production of amyloid disease. A radiograph taken after a few days of illness shows distinct periosteal thickening. In the chronic stage it reveals the softened area surrounded by new-formed thicker bone.

Prognosis.—This is very serious. The cases of most intense infection die with severe septic symptoms after 3 or 4 days illness, perhaps before a diagnosis can be made. If an abscess is suspected, incision promptly made, and the pus evacuated, the symptoms of sepsis disappear rapidly. The danger is, however, by no means over, since complicating pyemic conditions may arise later, or the patient die exhausted from the prolonged suppuration. In cases which survive the limb is liable to be shortened, and even amputation may have been necessary.

Diagnosis.—This is by no means always easy. Radiography will be of great aid in all doubtful cases. In the 1st year of life the symptoms may be supposed to be dependent upon some *gastrointestinal disturbance*. This is particularly true in infancy, if the location of the inflammation is in the neighborhood of some deeply seated joint, such as the hip; inasmuch as early redness and swelling are less likely to be exhibited here, and the age precludes any description by the patient of the seat of pain. In older subjects the possibility of *acute rheumatism* is the chief diagnostic problem. As a rule in this latter disease the inflammation is in the joint instead of the shaft of the bone; there is pain on movement of the joint itself; more than one articulation is affected; and the constitutional symptoms are less severe. *Erysipelas* gives rise to redness and swelling as in osteomyelitis, but there is no great tenderness or pain on movement, and no marked septic symptoms. *Scurvy* produces a painful and very tender swelling in the shaft of the bone, with great pain on movement, pallor, and some fever. There is, however, a total absence of the severe constitutional symptoms of osteomyelitis.

Treatment.—It is most important that treatment be instituted at the earliest possible moment, as otherwise irreparable damage or loss of life will result. It is entirely surgical in nature, and consists in incision of such sort that the pus shall be freely evacuated. This involves opening through the bone into the medullary cavity. Meanwhile the affected region is to be kept at rest. An effort may be made to modify the inflammatory process by the employment of autogenous vaccines. Later in chronic cases various other operative procedures may be required.

Arthritis

Only a résumé of this extensive subject can be given. The disease is often intimately associated with that of the bones; in some varieties being secondary to it; in others followed by an extension of the process to the bones from the joint. Inflammation of the joints associated with rheumatism or tuberculosis is described elsewhere. Rheumatoid arthritis and arthritis deformans have been grouped with rheumatism as a matter of convenience.

Acute Infectious Arthritis of Infancy (*Acute Infectious Epiphysitis*).—This disease is in cause and nature identical with the osteomyelitis described, differing chiefly in the early age and the seat of the lesion, the latter at the onset being either the epiphysis or the joint itself. Any of the ordinary species of pyogenic bacteria may be active, and the gonococcus or pneumococcus is sometimes the cause. They may enter the blood from various regions, not infrequently in young infants through the umbilicus or through some trauma sustained at birth. The disease occurs chiefly in the 1st year of life, and oftenest in the first 6 months. It may be mono-articular, or one articulation after another may suffer or several be attacked simultaneously. The joints oftenest involved are the hip and shoulder; the small joints being attacked less frequently. The onset is sudden, and the *symptoms* are indicative of sepsis. The swelling of the joints comes on rapidly, with pain, tenderness, redness, and rigidity, followed promptly by evidences of suppuration. In the severest cases death may occur in a few days, usually from some visceral septic complication. In others, less severe, suppuration develops more slowly with destruction of the articulation, an abscess appearing in from 2 to 3 weeks and discharging near the joint, or burrowing widely unless relieved by operation. The *diagnosis* in polyarticular cases is to be made especially from rheumatism; yet the early age of the patient almost excludes this disease. The *prognosis* is on the whole less unfavorable than in the osteomyelitis described, and the *treatment* similar to that for this disease.

Inasmuch as the forms and symptoms of acute arthritis often depend largely upon the germ which is active, it is sometimes convenient to classify the varieties of arthritis etiologically:—

Gonococcic Arthritis.—This occurs consecutively to a gonorrheal ophthalmia or vulvovaginitis, or it may be without discoverable portal of entry. It is not a very uncommon affection in early life, yet relatively infrequent as compared with the number of cases of gonorrhea occurring, especially vulvovaginitis. Gittings and Mitchell[1] found it but 3 times in 188 complications of gonorrheal vulvovaginitis reported by different observers. Its development in infants as a sequal to gonorrheal conjunctivitis was first pointed out by Cl. Lucas in 1885.[2] It begins acutely, often several weeks after the primary infection, or even after recovery from the symptoms of this; and may be single, but is frequently multiple, particularly in infancy. In the latter event it may exhibit the usual *symptoms* of the infectious arthritis as described, and the effusion be purulent. The wrists, knees, ankles, and small joints of the fingers and toes are those oftenest involved. In the less severe cases there is swelling from effusion, not much redness, slight pain, and moderate fever, and suppuration is not common. The process is generally limited to the synovial membrane. Recovery takes place after a number of weeks of illness, with or without final interference with the motility. *Treatment* consists in the employment of cold applications to relieve the pain, in putting the part at rest, and in evacuating pus should this be produced. The employment of gonococcus vaccines may be tried. (See Gonorrheal Vulvovaginitis, p. 222.)

Pneumococcic Arthritis.—This disease is an uncommon one, but occurs at least as frequently in early life as in adults. In an analysis of 100 collected cases made by Nitch[3] 31 were in children under 14 years of age, and 18 of them in the 1st year. Nattan-Larrier[4] records an instance in an infant of 3 weeks, developing consecutively to an operation for harelip. Although frequently associated with pneumonia, this is much less often the case in children than at a later period. The arthritis may develop after empyema or otitis, or may appear to be primary, the portal of entry not being discoverable. It is probable that the large majority of cases of suppurative arthritis in the first 5 years of life are pneumococcal (Dudgeon and Branson).[5]

The *lesions* are oftenest mono-articular, the part involved being the knee, shoulder, hip, elbow or ankle in the order named (Herzog).[6] Occasionally more than one joint is attacked, but a widespread septic polyarthritis is generally dependent upon other germs, or at least not upon the pneumococcus alone. The effusion may be serous only but is oftener purulent. The inflammation is usually less intense than when dependent upon other germs, and erosion of the cartilages does not occur so frequently.

The constitutional *symptoms* are not prominent unless a general septic infection develops. They consist chiefly of irregular fever and decided anemia. Locally there are the usual evidences of inflammation of a joint, yet without circumscribed redness. The swelling and edema may involve the whole limb. It would appear, however, that edema may fail to develop. The *prognosis* is dubious. Half or more of the cases die, although the outlook is considerably better in children than in adults, and the destructive changes in the joints are usually not great as compared with some other forms of suppurative arthritis. Pneumococcic complications in other parts of the body are the cause of death in many instances. Cases in which the pus is thick and greenish-yellow are more favorable than those with a dark-colored or watery purulent fluid.

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[1] Amer. Journ. Dis. Child., 1917, XIII, 448.

[2] Brit. Med. Journ., 1885, I, 429; II, 57.

[3] Brit. Med. Journ., 1907, II, 729.

[4] Arch. gén. de méd., 1905, CXCV, 528.

[5] Lancet, 1903, II, 316.

[6] Jahrb. f. Kinderh., 1906, LXIII, 462.

In the line of *diagnosis* this form of arthritis is to be distinguished from the gonococcic by the absence of evidences of gonorrhea elsewhere and the presence of wider extension of swelling and edema, should these be present. In acute articular rheumatism the inflammation is generally multiple, shifts from joint to joint, and is not purulent. Staphylococcic arthritis produces more decided symptoms; there is high fever, vomiting, marked redness about the joint, and a severe constitutional impression. Yet in all cases, a positive diagnosis of pneumococcic arthritis is to be made only by exploratory puncture and bacteriological examination. *Treatment* is entirely surgical. Incision is generally to be preferred to aspiration.

Arthritis Depending upon Other Causes.—Omitting from consideration the acute infectious arthritis of infancy, and the tuberculous and syphilitic lesions to be described later, arthritis may be due to a number of other causes than those mentioned. In some cases the process may perhaps be a toxic one, as, for instance, in the non-suppurative arthritis seen after some of the infectious diseases, especially scarlatina, and rarely typhoid fever, measles, mumps and other like disorders. A very frequent form is a mild arthritis, limited to the synovial membrane (*synovitis*) following trauma, such as a sprain or a contusion. In arthritis due to such causes there is pain, tenderness and swelling of the joints, but without any decided constitutional symptoms except in severe cases. Less often the arthritis may be suppurative and generally mono-articular. Rarely the meningococcus, colon-bacillus or influenza-bacillus may be found in the pus from the joints; but much the most frequent germ is the staphylococcus or streptococcus. The constitutional symptoms are severe, and the joint red, swollen and tender. The whole course is more intense than in pneumococcic arthritis. The prognosis is worst in the streptococcic cases. The treatment of the non-suppurative cases consists in placing the joint at rest and the application of cold. If pus has formed, the treatment is entirely surgical.

TUBERCULOSIS OF THE BONES AND JOINTS

Etiology.—Tuberculosis of these regions is of very frequent occurrence in early life, especially after infancy is passed. That of the bones is dependent upon some primary and often distant, large or oftener small and unrecognized, focus elsewhere in the body. The focus is often in the bronchial glands. Tuberculous arthritis is usually secondary to disease of the neighboring bones. The occurrence of some infectious or debilitating disease, or of a trauma, even slight, is a powerful predisposing cause. This is especially true of measles and pertussis. Patients, too, often show a familial predisposition to tuberculosis. Either variety of the tubercle-bacillus may be active, but, according to most investigators, it is more frequently the human type.

Pathological Anatomy.—The principal parts affected are the spinal vertebræ and the hip-joint, about ⅓ of the cases occurring in each region. In 8304 cases of tuberculosis of the bones and joints in subjects under 12 years reported by Bradford and Lovett[1] the distribution was: Spine 3840; hip 3192; knee 659; ankle 559; elbow 54. Other regions are attacked less frequently, among these being the tarsus, carpus, ribs, wrists, shoulder-joints and the bones of the skull. The upper extremities are much less often involved than the lower. In the former the elbow is the most frequent seat of the disease.

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[1] Orthopedic Surgery, 1915, 4.

The process in the bones is an osteitis beginning in the cancellous tissue of the bodies of the vertebræ; the bones of the carpus and tarsus; the central portion of the short tubular bones, such as the metacarpal bones and the phalanges; or the epiphyses of the long bones and extending thence to the joints; or the lesions may appear in the form of scattered nodules, as in the bones of the cranium. The first change is congestion of the vessels, followed by small-celled infiltration and the formation of miliary tubercles. These latter, with the granulation tissue produced, erode the bone and give rise to small cavities, which are filled with cheesy material containing bone-sand. This may be shut in and encapsulated and the disease arrested; or the process may continue and extend to the periosteum or into the joint and an abscess form, burrow in various directions, and finally rupture spontaneously or be relieved by operation. Suppuration then continues until all the diseased bone has been softened and discharged. Meanwhile a reparative action goes on, new bone being formed. Recovery finally takes place, although often with permanent injury to the bone remaining.

Tuberculosis involving a joint produces miliary tubercles and granulation tissue upon the synovial membrane, which is red and swollen and then ulcerated, and is finally separated from the cartilage leaving the bone exposed. A more or less purulent fluid accumulates and may discharge externally, or the process may be arrested earlier and heal without this. After recovery in the suppurative cases, ankylosis or deformity of the joints may remain. As already stated, tuberculous arthritis is generally secondary to infection of the bone, but sometimes, especially when the knee is involved, the process begins in the synovial membrane.

The recovery from tuberculous lesions of the bones or joints is a matter of months or much oftener years. In many cases there is never any external evidence of suppuration, although a joint may be severely affected and finally recover ankylosed. Generally only one joint is involved, but to this there are numerous exceptions. In the vertebral column one or several adjacent vertebræ suffer. Secondary tuberculous processes may develop, among the most frequent being meningitis. Amyloid degeneration of the liver, spleen and kidneys is a frequent development after prolonged suppuration.

Symptoms.—The onset of tuberculous disease of the joints or bones is insidious, and the symptoms often misleading. There is in general but little pain early in the case. The development of abscess is slow, without much fever, and with little constitutional disturbance. The early employment of radiography will often be of diagnostic aid. Further symptomatology is best discussed under the individual localizations of the tuberculous involvement.

1. TUBERCULOUS DACTYLITIS

This disease, often called "spina ventosa," is the chief representative of tuberculosis of the short tubular bones, other bones of this class being rarely affected. It occurs most frequently in early childhood, and involves the phalanges and the metacarpal bones of the hands and feet. One or more may be attacked. The most frequent seat is the phalanx of the index-finger. The centre of the bone undergoes destruction and the periphery is thinned and expanded, while the periosteum thickens in the regenerative process. The entire finger in consequence develops a spindle-shaped hard and, later, red swelling, the soft tissues sharing in the inflammation, and an abscess generally developing. Sometimes, however, the disease stops short of this. The course is comparatively painless, but long-continued, the process often ceasing only after many months of illness, and with much deformity or even loss of the finger. The diagnosis is to be made chiefly from syphilitic dactylitis. This occurs in younger subjects, and is often symmetrical and multiple, while other evidences of syphilis are present. The diagnosis is, however, frequently difficult. Treatment consists in improving the general health, particularly by all measures suitable for the management of tuberculosis in general. (See Vol. I, p. 561.) The part should be at rest with a splint or a plaster dressing applied. Operation should be performed only if abscess forms, in which case a free incision with the removal of dead bone will shorten the duration of the inflammation.

2. TUBERCULOUS SPONDYLITIS

(Caries of the Spine; Pott's Disease)

This is one of the most frequent tuberculous manifestations in early life. Although it may occasionally occur in infancy, its greatest incidence is between the ages of 3 and 14 years. More cases are seen between 1 year and 5 years than in any other 5-year period (Young).[1] It consists of a tuberculous osteitis beginning in the bodies of the vertebræ, producing caseous material and destroying the bone and spreading to all the tissues of the articulation. The spinous processes and arches are unaffected, and as a result kyphosis gradually develops as the bodies yield under the pressure of the weight of the upper part of the child, with some degree of scoliosis if the vertebræ softens more upon one side. One or often several vertebræ are involved, and the extent and form of the kyphosis depends upon the number (Figs. 385 and 386). The inflammation may result in abscess which may burrow in various directions; or may cease without the visible formation of pus, and bony union may take place. The dorsal region, especially the lower part, is the portion of the column oftenest attacked in early life; the lumbar much less frequently, and the cervical still less often. The pathological process has been described in the general discussion of tuberculosis of the bones.

Symptoms.—The onset is insidious and misleading, and varies with the region of the spine which is diseased. The earliest manifestations consist of restlessness, fretfulness and disturbed sleep; and of pain which may be persistent or intermittent, and occurs in the course of the spinal nerves which arise from the affected part. There may be pain in the back increased by pressure on the head or by jumping, but not when the pressure is applied over the seat of the lesion. There are also muscular rigidity of the back and the disposition of the child to assume some unusual position which will best take the weight from the diseased region and prevent jarring. Thus a child may avoid bending to reach an object on the floor; walk stiffly or carefully upon the toes; prefer to lie on the abdomen; rest frequently across a chair or over the mother's lap or with the hands on the thighs. In the cervical cases the child may hold the head very stiffly and even support it with the hands. Evidences of paralysis or other nervous disturbances often appear. These have been described under Compression Myelitis (p. 381). Paraplegia occurs in from 10 per cent. to 20 per cent. of cases of tuberculosis spondylitis. It is seen in about half of the cases of caries of the upper dorsal or cervical region, but is rare if the diseases is below the mid-dorsal spine. The average duration of the disease is 3 years before paralysis appears; but the symptom may develop even after a few months. Abscess may not occur at all, or may not appear until the 3d or 4th month or more after the onset, or oftener not until the 2d year of the disease. It is prone to develop insidiously and without acute symptoms (*cold abscess*). Abscess in cases of cervical caries often opens in the pharynx (*retropharyngeal abscess*), or may do so above the clavicle. In disease of the lower cervical and upper dorsal vertebræ it may burst into the pleura or make its way to the scapula, but often it gravitates to the lower part of the trunk and is discharged above Poupart's ligament. Lower dorsal and lumbar abscess often open above Poupart's ligament, in the loin, or in the upper portion of the thigh. The development of abscess occurs most frequently in involvement of the lumbar vertebræ. Kyphosis also is a late symptom of spondylitis. It begins as a slight curved projection over one vertebra, but increases in extent (Fig. 385). It is most visible in the mid-dorsal region, and the worst curvatures are found here. It may fail to develop in the lumbar and cervical regions. With kyphosis of the dorsal region, if well-marked, there is of necessity an alteration in the shape of the thorax, with flattening of the sides and prominence of the sternum (Fig. 386).

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[1] Orthopedic Surgery, 1906, 209.

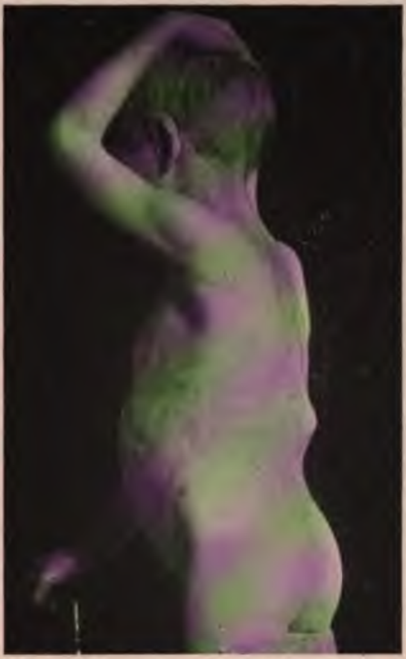

FIG. 385.—CARIES OF THE VERTEBRÆ. *Courtesy of Dr. I. Valentine Levi.*

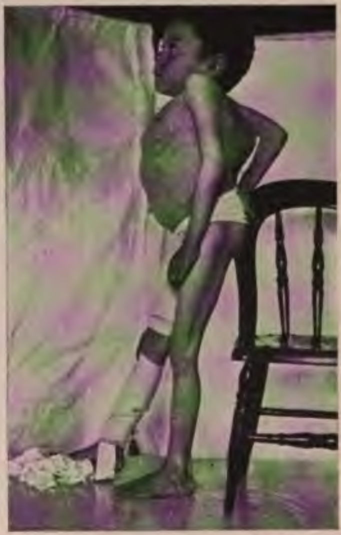

FIG. 386.—CARIES OF THE VERTEBRÆ. Advanced case with great angulation. *Courtesy of Dr. H. R. Wharton.*

Course and Prognosis.—The course of the disease is slow, with little or no fever, and abscesses forming are often long quiescent and undetected, and even when discovered for a long time show no tendency to rupture. The process may last from 2 to 5 years, unless recognized and treated early. If this early treatment is instituted the course is considerably shorter, and after 2 or 3 years the reparative process begins and recovery with ankylosis results with but slight deformity. Even many cases untreated until after paraplegia develops may finally regain the entire use of the limbs. There is always, however, great danger of relapse of a spondylitis, brought about by trauma or other cause. The prognosis in childhood is on the whole good, if treatment is systematically followed. Yet always the disease is a serious one, and many deaths occur from the result of prolonged suppuration, myelitis, or the development of a general tuberculosis. The mortality probably varies from 10 to 25 per cent.

Diagnosis.—It is most important that this should be made early. To depend upon the development of curvature for this is to allow the disease to advance unnecessarily far. When any of the symptoms mentioned are present, a careful, thorough examination of the child undressed should be conducted. The mobility of the spine; the position assumed; the gait; the distribution of pain; and the knee-jerks and other evidences of involvement of the spinal cord must be studied. Naturally the spinal column must be searched for beginning curvature, but before this has become evident the diagnosis should have been formed from the other symptoms. Radiographic examination is of value only late in the disease.

Certain other pathological conditions are especially to be distinguished from spinal caries in some of its stages. *Rachitis* produces a kyphotic curvature of the spine; but it is of much greater length and uniformity, is unaccompanied by rigidity, and disappears when traction is applied to the legs or the child suspended from the armpits or placed face downward and the legs elevated. (See Vol. I, p. 594, Figs. 204, 205.) This disappearance may not be true of long-continued cases of rickets, but in these other confirmatory rachitic symptoms will remove confusion. *Scoliosis* from rachitis or faulty position may, if slight, suggest the lateral curvature sometimes produced by caries. It is, however, of considerable length, while that of caries is short, and the former is not accompanied by rigidity or pain. *Hip-joint disease* may be supposed to be present in cases where the lameness is in reality the result of caries of the lumbar spine. The latter disease is without the limitation of movement at the joint, except in the direction of extension if a concealed psoas abscess be present; and under these circumstances the thigh is kept more or less flexed. The paralysis and other symptoms attendant upon spinal caries are to be distinguished from *nervous symptoms due to other causes.* This has been considered under Compression Myelitis (p. 380).

Treatment.—This is principally medical and mechanical rather than operative. The details will be found in works upon orthopedic and general surgery. The first great principle is the securing of absolute rest of the diseased spine. To accomplish this in the early acute stage the child must be kept recumbent upon a special bed and the affected region immobilized (Willard).[1] This should, however, be so arranged that the patient shall not be confined to the house. In about 3 months, after all such acute symptoms as pain and the like have disappeared, it is allowable to have the child fitted with a mechanical support and placed in an upright position. It is to be noted, however, that some orthopedic surgeons favor ambulatory treatment with mechanical apparatus from the beginning of the disease. Equally important is the maintaining and improving of the general health in every possible way, as in tuberculosis in any part of the body; life in the open air being one of the most important features.

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[1] Surgery of Childhood, 1910 424

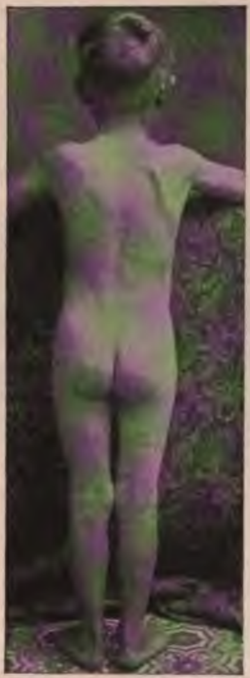

FIG. 387.

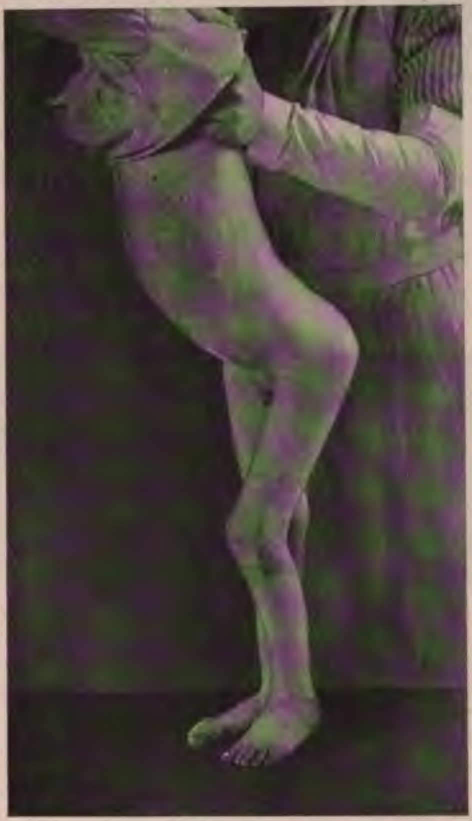

FIG. 388.

FIG. 387.—DISEASE OF RIGHT HIP-JOINT.

Internatal crease inclined toward left thigh. Right buttock narrower, gluteofemoral crease shortened and less distinct, buttock fading away into thigh. (*Willard, Surgery of Childhood*, 462, *Fig.* 406.)

FIG. 388.—HIP-JOINT DISEASE.

Shows flexion at hip and knee and outward rotation. (*Courtesy of Dr. H. R. Wharton.*)

HIP-JOINT DISEASE
(Tuberculous Coxitis)

This is the most common tuberculous disease of the joints in children, and 85 per cent. of the cases occur before the 10th year. In the statistics of Young[1] on 421 cases there were 5 in the 1st year, 132 from 1 to 5 years, and 136 from 5 to 10 years; 351 in all developing before the 15th year. Rather more males than females are attacked. Hip-joint disease nearly or quite equals spinal caries in frequency. In 1000 cases of tuberculous disease of the bones collected by Young,[2] the vertebræ

[1] Orthopedic Surgery, 1906, 302.
[2] *Loc. cit.*, 301.

were affected in 41.6 per cent. and the hip in 42.1 per cent. In the large majority of instances only one hip is involved, and, if both, then not simultaneously. Sometimes the disease begins in the synovial membrane (20 per cent. to 30 per cent., Spitzky);[1] but, as a rule, an ostitis of the epiphysis of the femur is the first process, followed later by arthritis in which all parts of the joint share, and still later by the formation of abscess and destruction of the head of the femur, displacement, and deformity. A reparative process may begin at any time.

Symptoms.—The onset is usually gradual, the first symptom being a slight lameness, which is generally intermittent and seen oftenest on first rising in the morning or after exercise. Days or weeks may pass without any return of lameness being discoverable. Some degree of pain may also be present, now or developing later, usually referred to the front of the knee or the inner side of the thigh. There is a disposition, too, to stand with the weight of the body thrown upon the sound leg, the other being abducted and rotated outward. When the child is standing with the feet close together, there is a flattening of the gluteo-femoral fold on the diseased side, and a deviation of the internatal crease toward the healthy side (Fig. 387), and when placed on the back on a table with the legs together, the diseased side appears longer. Distinct muscular rigidity is shown if efforts are made to move the diseased hip. At first this is seen only during backward extension with the child on the abdomen or side, but later motion in any direction is restricted, and, with the patient on his back, the pelvis may be found to move with the leg, if the fingers are placed upon the anterior superior spinous process of the ilium. Swelling and tenderness about the joint may be detected even early in the course by feeling for the head of the femur under Poupart's ligament close to the femoral artery. Atrophy of the thigh begins early.

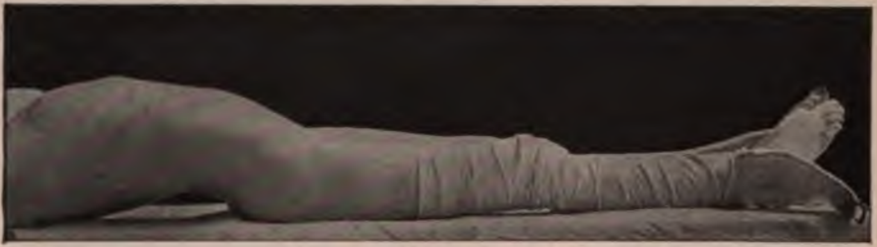

FIG. 389.—HIP-JOINT DISEASE.

When knee is brought down to table, the lumbar spine is arched, showing fixed flexion of thigh and consequent lifting of pelvis. (*Willard, Surgery of Childhood*, 463, *Fig.* 408.)

As the disease advances there is an increase of the limp, with pain on walking; and there are startings and "night cries" from the occurrence of pain as the result of involuntary movement during sleep. The suffering from movement may become so decided that walking is impossible, or accomplished only with a very marked limp; and even when at rest the pain is often very constant and intense. The thigh is now held partially flexed and rotated outwardly, and the knee flexed (Fig. 388). When the child is on its back upon a table, with the legs extended and parallel, the diseased leg now exhibits an apparent shortening, owing to tilting of the pelvis upward, and the back is arched (Fig. 389). The atrophy of the thigh and leg become decided.

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[1] Pfaundler and Schlossmann, Dis. of Child., Amer. Trans., 1912, V, 222.

Destruction of the joint proceeds, with the production of pus within it; the thigh is flexed, adducted, and rotated inward; the knee is flexed; the swelling about the hip increases, and an abscess may form with fluctuation and final discharge. This evacuation is oftenest immediately in front of the joint, but burrowing in other directions may occur before an opening forms. It is to be noted, however, that absorption of the head and neck of the femur may take place without any visible evidence of suppuration. Statistics upon the frequency of abscess show a great variation. The probability of its occurrence depends to a large extent upon how early appropriate treatment was commenced. A study by Young[1] of various published series gives the proportion of abscess as from 33 per cent. to over 50 per cent. of the cases. Actual shortening of the limb, often of several inches, is now present, shown by measurement from the anterior superior spinous process to the internal malleolus, resulting from the destruction of the head of the bone and the erosion of the acetabulum, combined with diminished growth of the femur in length. A very marked lordosis of the spine develops.

Constitutional symptoms are varied. In the early stages there are restlessness, fretfulness, and sometimes slight rise of temperature at night. Later when pus is forming, the fever may be more decided, but may fall when discharge takes place.

Course and Prognosis.—The course is prolonged. It is usually 3 or 4 months, although sometimes less, before the osteitis extends to actual involvement of the joint and the severe symptoms begin; and from 2 to 4 years, and still longer in cases with abscess, before the whole process is over and the symptoms of inflammation have entirely disappeared. The more promptly treatment is commenced the shorter in general will be the attack. It is possible in many cases in which treatment is begun in the first few weeks for the process to cease entirely before the joint itself is attacked, and for recovery to be complete without stiffness or any alteration of the gait. The large majority, however, are not so fortunate, and more or less permanent lameness persists. There is generally not much shortening remaining, unless there has been a destructive process within the joint. If proper treatment has not been employed, many children recover with the hip-joint ankylosed in a faulty position.

Even years after recovery recurrences may readily and repeatedly occur from some slight trauma; and exercise of quite moderate nature, such as long walking, dancing, and the like, may be sufficient to occasion this. The disease may end fatally from the results of prolonged suppuration, or from the development of tuberculosis elsewhere in the body, especially general tuberculosis or meningitis.

The prognosis on the whole is good, both for life and for the possessing of a fairly useful limb, especially in children when the diagnosis was made early and in whom the general health is good. It is difficult to determine the death-rate, since the circumstances under which various statistics have been gathered differ considerably. To come to any accurate conclusion the after-history of the patient should be followed for a number of years. Probably from 10 per cent. in patients treated early to 60 per cent. in neglected cases already suppurating would fairly express the final mortality (Willard).[2]

Diagnosis.—This should be made as early as possible and is based upon a consideration of the symptoms described, including careful examination of the child undressed, walking, standing and recumbent. Especially important are the gradual development of a limp; the reflex pain; the position of the limb; and the muscular rigidity as elicited by passive movements. Radiography is of great aid, the joint early appearing indistinctly outlined, and in advanced cases showing undoubted evidences of destruction of the bone and of faulty position. *Rheumatism* is often suspected, but this is seldom confined to the one joint, and is of acute onset with fever and with localized swelling and pain. *Spinal caries* may produce flexion of the thigh from the production of a psoas abscess. The restriction of movement is generally, however, limited to extension. *Trauma* of the hip causes acute local pain, swelling, lameness, and even suppuration; but the onset is acute after injury and there is no muscular rigidity. An *infectious arthritis* is acute in nature and rapid in its course. The referred pain of hip-joint disease may suggest the existence of *tuberculous arthritis of the knee-joint*, but examination will readily show that there is no inflammation there. *Juvenile osteochondritis deformans* (Perthes' Disease)[1] is a rare condition producing symptoms very similar to those of tuberculous coxitis. In 1500 cases of disease of the hip Delitala[2] found only 6 of this condition. It is distinguished from tuberculous disease of the hip to a certain degree by the lack of pain and spasm; the limitation of motion chiefly on adduction; the absence of evidences of rarefaction of the bone on *x*-ray examination; and the negative von Pirquet reaction. Yet often the diagnosis cannot be made early in the case. The difficulty in diagnosis of hip-joint disease even in the early stages depends chiefly on lack of care on the part of the examiner. In the later stages a mistake is not often possible.

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[1] *Loc. cit.*, 317.

[2] Surgery of Childhood, 1910, 496.

Treatment.—The treatment consists in efforts at aborting the inflammation, or allaying it if too far advanced for this. This is to be accomplished through putting the part entirely at rest and removing the influence of muscular spasm by means of traction. Whether this shall be done with the child in a recumbent position in bed, or by means of a suitable mechanical ambulant apparatus, depends upon the character of the symptoms, the social position of the child, and the views of the surgeon. It is a question requiring so much care and detailed knowledge that no one but a surgeon should attempt to solve it. The same remark is true of the management of abscesses which may occur, and of the determining the nature of and need for other operative treatment. In addition to these surgical measures, it is of the greatest importance to have the child placed under hygienic conditions the most favorable possible, and to maintain and improve the general health in every other way. Open-air life, preferably at the sea-shore, is a great desideratum.

4. TUBERCULOUS ARTHRITIS OF OTHER JOINTS

Less frequently tuberculosis may affect other joints than the hip, oftenest the knee, next the ankle, and after this in childhood the elbow. (See p. 440.) The nature of the pathological process has already been described. In the elbow the lesions are oftenest first situated in the end of the humerus, and only exceptionally in the radius alone. In the ankle the disease appears usually first in the bones of the tarsus, less frequently in the end of the tibia (Fig. 390). In the knee it begins in one of the condyles of the femur, and less often in the head of the tibia, or may develop first of all in the synovial membrane. One or more joints may be involved

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[1] Verhand. d. Gesellsch. f. Chir., 1913, XLII, II, 140.

[2] Rivista di clin. pediatrica, 1915, XIII, 426.

Symptoms.—These are analogous to those of hip-joint disease. The onset is slow. Pain may be severe, or often slight or absent early in the case; but later it is constant. Rigidity of the muscles is characteristic, and the joint is swollen and held in a flexed position. Redness is not an early symptom. Atrophy of the muscles develops, and high fever is present if suppuration occurs. The symptoms vary naturally with the location. In tuberculous arthritis of the knee (*white swelling*) the first manifestations are the swelling (Fig. 391) and an occasional slight limp, with a tendency to keep the knee flexed. The child walks on the toes, and the leg is adducted and rotated outward. If the ankle is affected there is likewise a limping gait, with abduction of the leg and eversion and outward rotation of the foot. In disease of the elbow-joint there is pronation of the arm with slight flexion of the elbow.

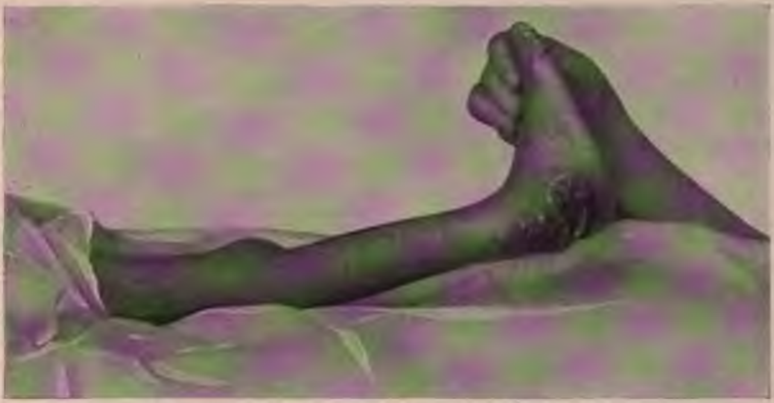

FIG. 390.—TUBERCULOSIS OF THE ANKLE-JOINT.
Courtesy of Dr. H. R. Wharton.

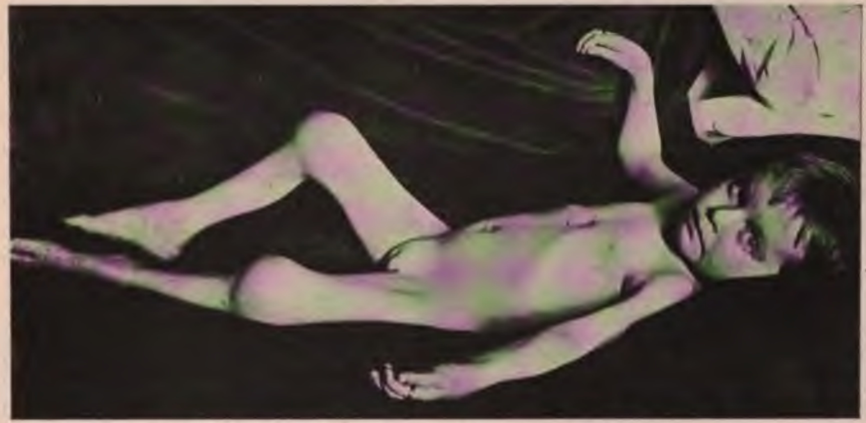

FIG. 391.—TUBERCULOUS ARTHRITIS OF THE KNEE-JOINTS.
Courtesy of Dr. H. R. Wharton.

The course is very similar to that of hip-joint disease. but the prognosis for life is better, although death may occur from the development of tuberculosis elsewhere, or from the results of prolonged suppuration. The diagnostic distinctions from other diseases are in general similar to those discussed under Hip-joint Disease (p. 446), and the same principles of treatment prevail.

SYPHILITIC DISEASE OF THE BONES

(See also Syphilis, Vol. I, p. 562)

Although a syphilitic arthritis is occasionally seen (see Vol. I, p. 576 and Fig. 193) the forms in which syphilis usually shows itself in the bony system are epiphysitis, dactylitis, and chronic osteoperiostitis.

1. SYPHILITIC EPIPHYSITIS

Etiology.—This affection, although recognized earlier, is often called "Parrot's Disease" from his description of it in 1872.[1] It is an early manifestation of hereditary syphilis, appearing generally within the first 3 months, and found even in infants still-born. It is the most common osseous manifestation of hereditary syphilis at this period.

Pathological Anatomy.—There develops an inflammation in the cartilaginous layer of the epiphysis next to the shaft and in the periosteum over this, with thickening of the epiphyseal plate, irregular proliferation of cartilage and necrotic changes. As a result the cartilage softens and loosens, and the epiphysis may separate; or, without separation, there may rarely be a secondary infection and suppuration may take place, and the joint may occasionally become involved (see Fig. 189, p. 572). The process is a gummatous infiltration of the subperiosteal tissue. The epiphyses of the large joints are oftenest affected, especially those of the humerus, usually on both sides; but a number of the long bones of the body may be attacked simultaneously.

Symptoms.—The symptoms develop acutely and are those suggestive of flaccid paralysis, with swelling and tenderness in the neighborhood of the epiphysis affected. The title "Syphilitic Pseudoparalysis" has frequently been applied to the condition.

The affected limb is not moved voluntarily at all, and passive motion causes an outcry. The degree of swelling is generally moderate, depending upon the part involved. At the shoulder it is often scarcely visible. Occasionally crepitus can be elicited. There is little or no fever. Radiography may show separation of the epiphysis.

Course and Prognosis.—The prognosis is favorable if the condition is not widespread, and if energetic treatment is promptly commenced. The swelling and tenderness rapidly disappear; even an epiphysis which has separated will reunite; and the normal condition of movement is regained in 2 or 3 weeks, or longer in more severe cases. There may be spontaneous recovery without treatment. Should, however, there be many epiphyses involved with severe syphilitic manifestations of other nature, or should suppuration of a joint take place or an osteomyelitis develop, the prognosis is unfavorable and the case will probably end fatally.

Diagnosis.—This rests especially upon the symptoms described, evidences of syphilis elsewhere in the body, the early age, and the Wassermann reaction. Although the condition suggests a paralysis, examination shows no alteration of the electrical response, while the presence of swelling, tenderness and pain on movement exclude the possibility of an *obstetrical paralysis* of the upper arm type, which is naturally suggested if the upper extremity of the humerus is the part affected. This latter condition is, moreover, present from birth. *Scurvy* exhibits many of the symptoms, such as swelling, pain and pseudo-paralysis; but rarely occurs so early in life, is not limited to the epiphysis, and is very promptly cured by anti-scorbutic treatment. *Acute infectious arthritis of infants* is attended by severe septic symptoms. *Rickets* is later in developing and has no such marked pain on movement; although there are, it is true, epiphyseal enlargement and often pseudo-paralysis.

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[1] Arch. d. phys. norm. et path., 1871–2, IV, 319.

Treatment.—The general treatment has already been described in the chapter on Syphilis (Vol. I, p. 581). In addition it is necessary to keep the part at rest in order to prevent pain. Surgical aid will be needed if suppuration develops.

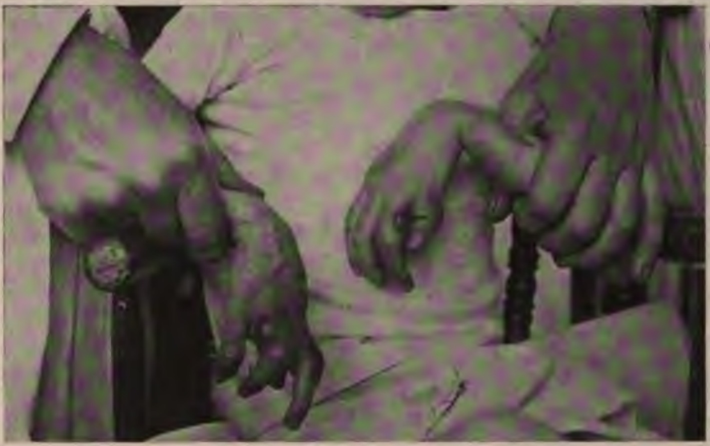

FIG. 392.—SYPHILITIC DACTYLITIS.
Courtesy of Dr. H. R. Wharton.

2. SYPHILITIC DACTYLITIS

This not common manifestation of syphilis is seen usually in the first 2 years of life, may be single or multiple; and is often symmetrical. It affects the phalanges more frequently than the metacarpal and metatarsal bones, being oftenest situated in the proximal phalanx of the index-finger. The lesion probably begins as a gummatous periostitis, an epiphysitis, or an osteomyelitis. The swelling usually does not go on to suppuration, and does not involve the joints. The symptoms do not differ materially from those of tuberculous dactylitis (p. 441); there being a red, hard, painless, fusiform swelling of the affected part (Fig. 392). It differs from the tuberculous disorder in being oftener multiple; less likely to attack the metacarpal and metatarsal bones; and of much less frequent occurrence. The prognosis, too, is better, in that in the earlier stages antisyphilitic treatment may be of service.

3. CHRONIC SYPHILITIC OSTEOPERIOSTEITIS

This is a manifestation of late hereditary syphilis, seen oftenest in later childhood, although it may occasionally occur in infancy.

Pathological Anatomy.—The lesions consist in the development of nodules upon the bones or of gummata within them, or of hypertrophic periosteitis with the formation of new bone and periosteal thickening. They may occupy several bones, generally symmetrically; those involved oftenest being the shaft of the tibiæ; the bones of the arm, and the cranium. The hyperplasia produces often great increase in the thickness of the long bones, as well as in their length to some extent. In other cases the enlargement is nodular and situated chiefly near the epiphysis. Gummatous deposits beneath the periosteum or within the bones may produce necrosis and softening, with suppuration and the formation of sinuses.

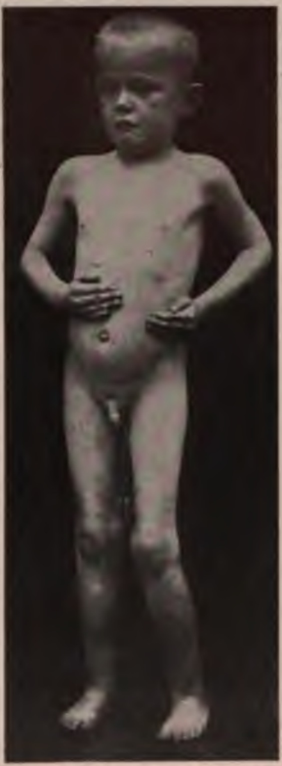

FIG. 393.—CHRONIC SYPHILITIC OSTEO-PERIOSTITIS.

Child of 11 years in the Children's Ward of the Hospital of the University of Pennsylvania. Head brachycephalic, occipital region almost perpendicular, frontal tuberosities, flattening of the bridge of the nose and typical sabre-tibiæ, spleen enlarged.

Symptoms.—The symptoms develop slowly and are very characteristic. There is a variable degree of pain, especially at night, with tenderness and a gradually increasing deformity. In characteristic cases the tibiæ on both sides are much thickened, often exhibit irregular nodules, and are bowed anteriorly and flattened on the sides (*sabre-tibiæ*) (Fig. 393). Other long bones show similar deformity. If the cranium is involved there are large irregular hyperostoses, especially of the frontal and parietal regions making the forehead square and projecting or upright (Fig. 393). The course of the affection is very chronic, and the disease is resistant to treatment unless this is commenced early. Any production of pus which may occur is of slow development and marked by little pain or constitutional disturbance, and the sinuses which result are long in healing, keeping open until the diseased bone has been softened and discharged as a sequestrum. The acute symptoms may be relieved by antisyphilitic treatment, but the bony thickening changes but little.

Diagnosis.—This is usually easy inasmuch as there is no disorder of early life which simulates this with the exception of *rickets*. The latter disease may produce anterior curvature of the tibiæ and bosses upon the cranium; but the curvature is situated generally near the lower epiphysis rather than in the middle of the shaft. There is also observed in *syphilis* the presence of pain, while in rickets other symptoms characteristic of that disease are present. The age of development of the syphilitic condition is much later. *Tuberculosis* may produce necrosis and sinuses of long duration, resembling those of syphilis, but th [illegible] tuated more often in the ends of the bones instead of the shaft.

Treatment.—This is that usual for the later symptoms of hereditary syphilis, consisting of the combination of mercury and the iodides.

MORBID GROWTHS OF THE BONES

Multiple Exostoses.—These are not very uncommon and may be single or more frequently multiple and more or less symmetrically arranged. Heredity is an etiological factor of considerable importance, and males are oftener affected than females. There is no evidence that any other disorder exercises a positive etiological influence in producing this. The exostoses are covered externally by cartilage while still growing. Under this is a layer of compact bone incasing cancellous tissue, with a central medullary cavity within, which communicates with that of the bone from which the growth springs. The growths are of a benign nature, and vary much in size and form. They are situated chiefly upon the long bones of the limbs, oftenest on the shaft near the epiphyseal junction, being especially likely to appear in the neighborhood of the knee, shoulder and wrist. Although they may be present at birth, they appear usually first during childhood and increase in number until early in adult life, after which time they may diminish in size. They may be few or very numerous. As a rule they are painless unless by pressure upon nerves; and they may interfere with motion to a limited extent. In this respect the disease differs from myositis ossificans, as also in the fact that there are no bony deposits in the muscles. Treatment by removal is necessary only when decided symptoms are produced.

Multiple Enchondromata.—Like exostoses these growths are often hereditary or familial. Rickets and trauma have possibly some influence in their production. They differ from exostoses in their histological structure, since they are composed of cartilage; and in their situation, as they occur oftenest upon the hands, fingers, feet and toes. They are prone to produce thinning of the bone and to undergo a cystic degeneration, but not to suppurate.

Malignant Growths.—*Primary* new growths of this nature situated in the bones are not of common occurrence in early life, except toward the end of later childhood when they become not infrequent. The most common is sarcoma, which has occasionally been seen even in infancy. Early in the case it is most liable to be mistaken for tuberculosis; but it is of rapid growth and very malignant, exhibits greater pain, and less often spreads to the joints. *Secondary* malignant neoplasms of the bones through metastasis may be of various sorts, and are much less often seen than in adult life. Sarcomatous tumors may develop in the bones of the skull, secondary to disease of the suprarenal glands. (See p. 526.)

A form of sarcomatous growth designated **chloroma**, from the green color on section, is a rare disorder at any time of life, but is sometimes seen in children. It develops in the bone-marrow, especially of the skull. With the other symptoms due to its local action, there is a condition of the blood similar to that present in lymphocytic or sometimes myelocytic leukemia; loss of strength; emaciation; fever; hemorrhage from the mucous membranes; enlargement of the lymphatic glands; and moderate enlargement of the spleen. The duration of the disease is 1 or 2 months. Metastases may take place into various lymphatic tissues of the body. Although so closely allied to leukemia in its symptoms, it seems better to regard chloroma as a primary neoplasm of the bones.

SECTION X

DISEASES OF THE BLOOD, SPLEEN AND LYMPHATIC GLANDS

CHAPTER I

DISEASES OF THE BLOOD

Our knowledge of the interrelationship of the various disorders of the blood to each other, and to the maladies of the different organs with the physiology and pathology of which they are often so intimately associated, is undergoing constant change under the influence of continued investigation. Consequently, any classification adopted must be open to criticism. The characteristics of the blood under normal conditions in infancy and childhood have been described to some extent in the section upon the Physiology of Early Life (Vol. I, p. 58). The fuller synopsis which follows has been very kindly written for me by Dr. Frank B. Lynch, Jr., Dickson Fellow in Bacteriology to the William Pepper Laboratory of Clinical Medicine of the University of Pennsylvania. It describes the appearances of the various formed elements of the blood, and states under what conditions they may be expected to be present, and whether in altered numbers. Under his supervision, too, the colored plate has been prepared, illustrating the cells of the blood, both physiological and pathological (Fig. 394).

NORMAL AND PATHOLOGICAL BLOOD CORPUSCLES

Erythrocytes.—**Number.**—About 5,500,000 per c.mm. for the first 2 years of life; almost 6,000,000 for the next 3 or 4 years; then falling to 4,500,000 or 5,000,000 toward puberty.

Size.—Diameter varies from 3.5 mikrons to 10.5 mikrons.

Appearance.—Round, regular, biconcave discs (1, in plate), staining with acid stains (eosin, etc.), and showing a central pale area surrounded by a deeper stained periphery. Rouleaux (2, in plate) of red cells are commonly described as being stacked, like coins. They are common in fresh, unstained specimens, but are rarer in smears.

Microcytes (3, 4, 5, in plate).—Erythrocytes, small in comparison with the average red blood-cell in the smear. They often show crenation and poikilocytosis.

Macrocytes (6, 10, in plate).—Erythrocytes, large in comparison with the average red blood-cell. They often show granular degeneration and polychromatophilia.

Anisocytosis.—Variations in size of the erythrocytes.

Poikilocytes (11, 15, in plate).—Irregular pear-shaped forms.

Crenated Cells (16, 17, in plate).—Cells which have given up some of their fluid to the serum or diluting fluid, and are wrinkled in consequence.

Granular Degeneration (9, 10, 24, in plate), or basophilic degeneration. The appearance of basic (blue) dots throughout the red cell,

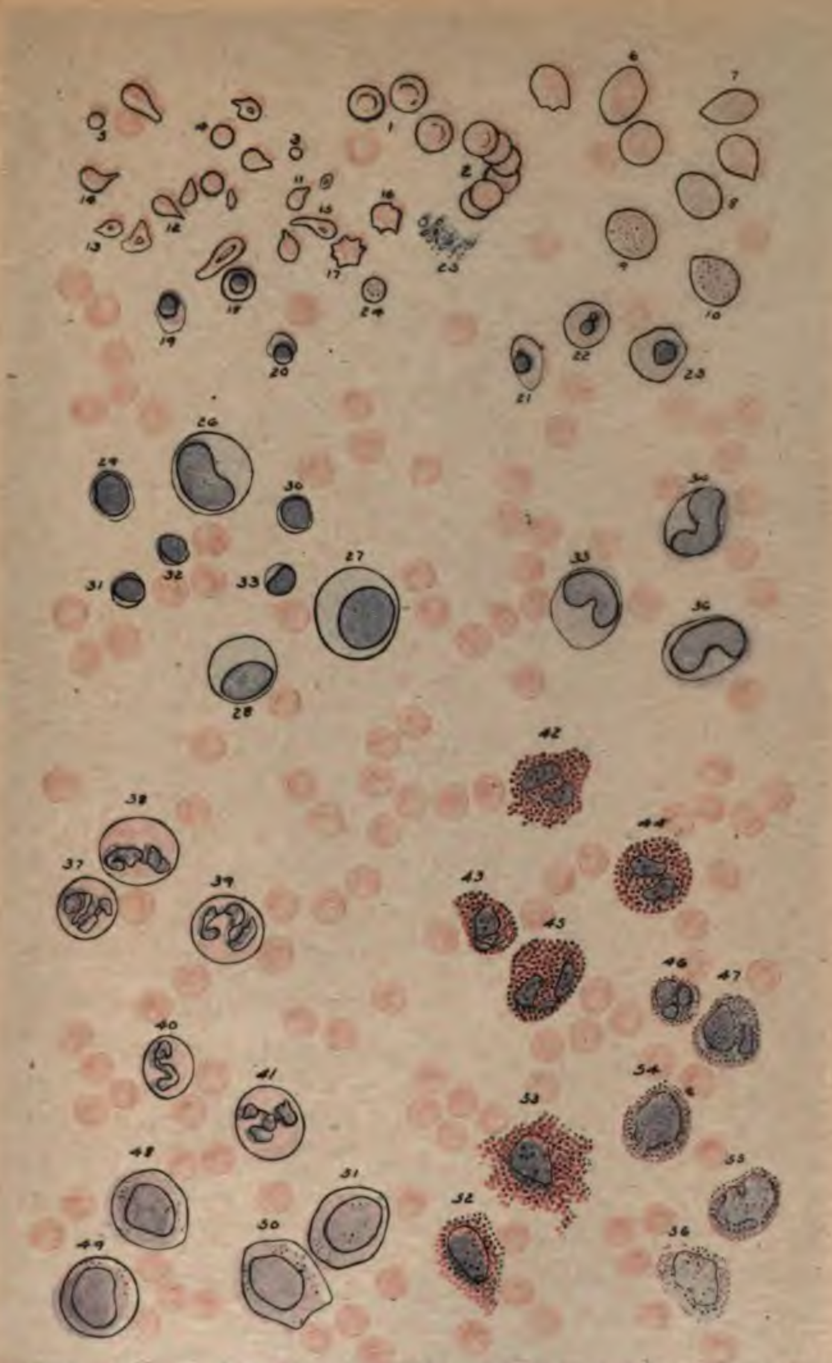

Fig. 394.—Varieties of Erythrocytes and Leucocytes in Normal and Pathological Conditions. (Wright's Stain.)

Explanation of the plate of Blood-cells in Fig. 394. (Wright's method of staining employed.)

Erythrocytes: 1, Normal erythrocytes; 2, rouleaux of erythrocytes; 3, 4, 5, microcytes; 6, 7, 8, 9, 10, macrocytes; 7, 8, 9, 10, show polychromatophilia; 9, 10, granular degeneration; 11, 12, 13, 14, 15, poikilocytes; 16, 17, crenated red blood-cells; 18, normoblast; 19, normoblast extruding nucleus; 20, microblast; 21, 22, 23, megaloblasts; 24, granular degeneration (see also 9, 10); 25, blood platelets.

Leucocytes: 26, 27, 28, Large mononuclear leucocytes; 29, intermediate type, classed as small lymphocytes; 30, 31, 32, 33, small lymphocytes; 34, 35, 36, transitionals; 37, 38, 39, 40, 41, polymorphonuclear neutrophiles; 42, 43, 44, 45, eosinophiles; 46, 47, basophiles (mast cells); 48, 49, 50, 51, neutrophilic myelocytes; 52, 53, eosinophilic myelocytes; 54 55, 56, basophilic *myelocytes.*

SECTION X

DISEASES OF THE BLOOD, SPLEEN AND LYMPHATIC GLANDS

CHAPTER I

DISEASES OF THE BLOOD

Our knowledge of the interrelationship of the various disorders of the blood to each other, and to the maladies of the different organs with the physiology and pathology of which they are often so intimately associated, is undergoing constant change under the influence of continued investigation. Consequently, any classification adopted must be open to criticism. The characteristics of the blood under normal conditions in infancy and childhood have been described to some extent in the section upon the Physiology of Early Life (Vol. I, p. 38). The fuller synopsis which follows has been very kindly written for me by Dr. Frank B. Lynch, Jr., Dickson Fellow in Bacteriology to the William Pepper Laboratory of Clinical Medicine of the University of Pennsylvania. It describes the appearance of the various formed elements of the blood, and states under what conditions they may be expected to be present, and whether in altered numbers. Under his supervision, too, the colored plate has been prepared illustrating the cells of the blood, both physiological and pathological (Fig. 394).

NORMAL AND PATHOLOGICAL BLOOD CORPUSCLES

Erythrocytes. *Number.*—About 5,500,000 per c.mm. for the first 2 years of life, almost 6,000,000 for the next 3 or 4 years; then falling to 4,500,000 or 5,000,000 before puberty.

Size.—Diameter varies from 3.5 mikrons to 10.5 mikrons.

Appearance.—Round, circular, biconcave discs (1, in plate), staining with acid stains (eosin, etc.), and showing a central pale area surrounded by a deeply stained periphery. Rouleaux (2, in plate) of red cells are commonly described as being stacked, like coins. They are common in fresh, unstained specimens, but are rarer in smears.

Microcytes (3, 4, 5, in plate).—Erythrocytes small in comparison with the average red blood-cells in the smear. They often show crenation and poikilocytosis.

Macrocytes (6, 10, in plate).—Erythrocytes, large in comparison with the average red blood-cell. They often show granular degeneration and polychromatophilia.

Anisocytosis.—Variations in size of the erythrocytes.

Poikilocytes (11, 15, in plate).—Irregular pear-shaped forms.

Crenated Cells (16, 17, in plate).—Cells which have given up some of their fluid to the serum or diluting fluid, and are wrinkled in

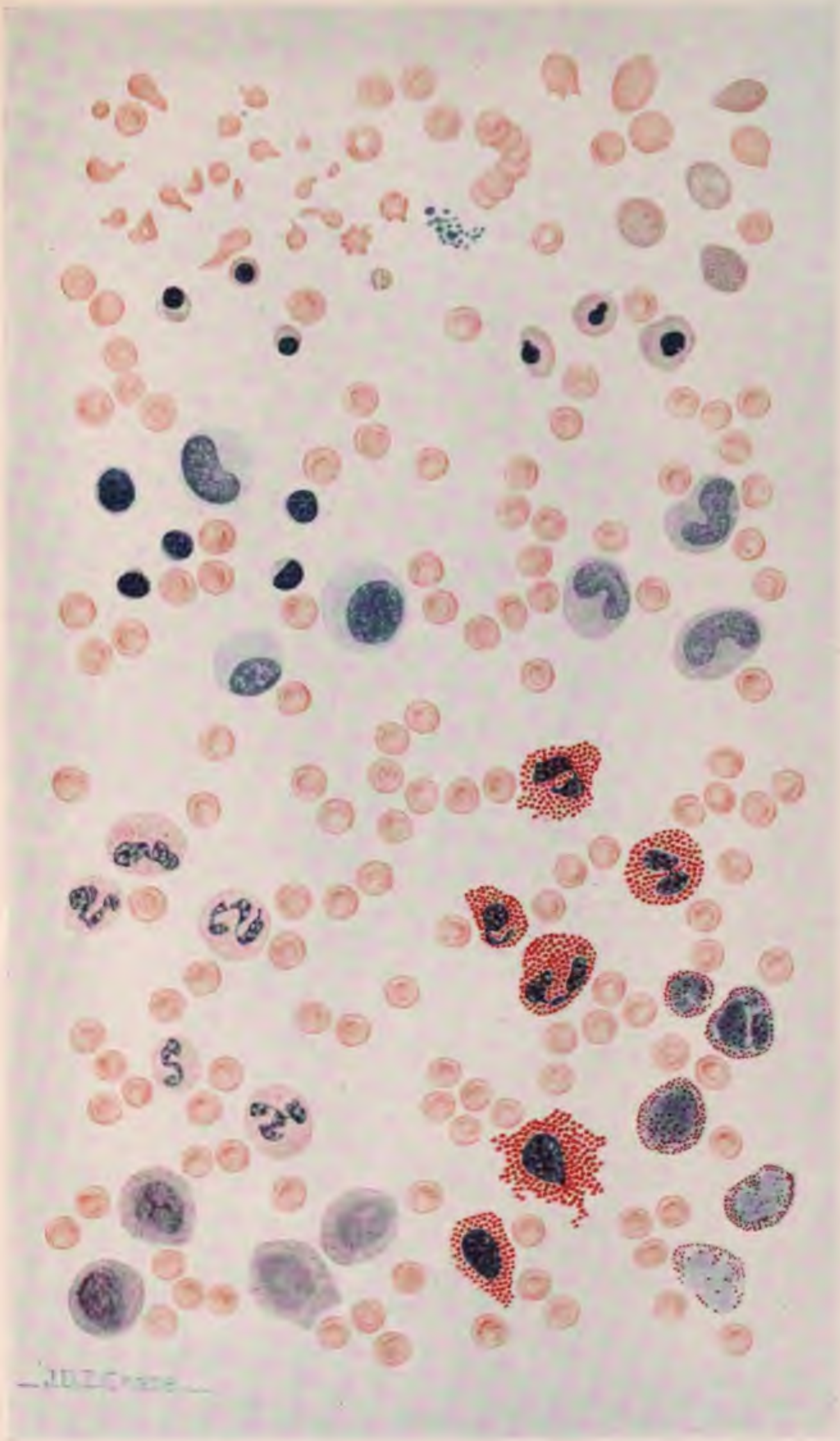

FIG. 394.—VARIETIES OF ERYTHROCYTES AND LEUCOCYTES IN NORMAL AND PATHOLOGICAL CONDITIONS. (WRIGHT'S STAIN.)

which may be otherwise normal, or may be nucleated, or may show polychromatophilia.

Polychromatophilia (7, 10, in plate).—A diffuse basic (bluish) tint throughout the red blood-cell.

Anisocytosis and poikilocytosis may occur in any anemia, even if mild in type. Granular degeneration and polychromatophilia may be seen in any severe anemia, but especially in lead-poisoning and in pernicious anemia. Crenated cells may be found, to a slight extent, even in normal blood.

Nucleated Red Cells (18, 19, 21, 22, 23, in plate).—These never occur in normal blood except in the 1st year of life, when an occasional normoblast or megaloblast may be found after careful search. They are present in any severe secondary anemia, in chlorosis, and in primary pernicious anemia.

Normoblast (18 in plate).—A nucleated red blood-cell of the same size as the normal erythrocytes in the smear.

Microblast (20 in plate).—A nucleated red cell smaller than the normal cells.

Megaloblast (21–23 in plate).—A nucleated red cell larger than the normal cells.

Nucleated red cells have a deep-blue nucleus, which may be round, oval, dumb-bell shape, or mitotic.

Blood Platelets (25, in plate).—Small, basic-staining bodies, occurring separately or in clumps. They are a constituent of normal blood.

Leucocytes.—The leucocytes of normal blood are lymphocytes, large mononuclears, transitionals, polymorphonuclear neutrophiles, eosinophiles and basophiles.

Myelocytes are leucocytes found only in abnormal blood, except in the first few days of life. Unstained leucocytes are white or cream-color.

Number.—In the new born 15,000 to 20,000 per c. mm.; in the 1st year and up to the 6th year 7000 to 16,000 per c.mm.; from the 6th year to puberty 7000 to 12,000 per c.mm. The variations in infancy and childhood are much greater than in adult life.

Lymphocytes (29–33, in plate).—These are small cells with a single nucleus. They range from the diameter of normal red cells to considerably larger in the younger forms. The nucleus is generally round or oval and takes the basic stain. The protoplasm is small in amount, often being present as a thin rim of blue staining-material around the nucleus. In the larger forms the protoplasm is larger in amount in comparison with the nucleus. Up to the 9th or 10th month the lymphocytes form about 50 to 60 per cent. of the leucocytes; then they decrease to about 55 per cent. during the 2d year, falling gradually to about 40 per cent. in the 6th year. By the 10th year they are about 30 per cent., which percentage they maintain until about puberty, after which they fall to the percentage present in the adult. Lymphocytosis is found in lymphocytic leukemia, pertussis, and syphilis. A relative increase is seen in measles and mumps.

Large Mononuclears (26–28, in plate).—These cells are about 2 or 3 times the diameter of normal red cells. The nucleus is round or oval, often with an indentation in the side. The cell-body often exhibits a cellular network, which gives somewhat the appearance of true granulations. The large mononuclears constitute 1 or 2 per cent. of the leucocytes. Large mononuclear leucocytosis is said to be present in malarial cachexia, measles, and in myelocytic leukemia.

Transitionals (34–36, in plate).—Cells of this type are about twice the size of normal red blood-cells. The nucleus is large, with a deep in-

Myelocytes are not found in normal blood after the first weeks of life. They occur in myelocytic leukemia; disease of the bone-marrow; pernicious anemia; acute lymphocytic leukemia; anemia pseudoleukemica infantum; small pox; severe diphtheria and severe septic infection.

Eosinophilic myelocytes (52, 53, in plate) are rarely found except in myelocytic leukemia, cases of tumor of the bones with involvement of the marrow, and in anemia pseudoleukemica infantum.

Basophilic myelocytes (54–56, in plate) are rare even in myelocytic leukemia.

LEUCOCYTOSIS

This title is employed to designate an increase in the proportion of white blood-cells, which may be either physiological or pathological. It is normally present in the new born, and after cold baths, exercise, and massage. With relation to the ingestion of food, a careful study by Mitchell[1] in 50 bottle-fed babies showed inconstant results, and oftener a diminution in the number of leucocytes than an increase after taking nourishment. A pathological leucocytosis generally of the polymorphonuclear cells is encountered in a large number of conditions. This has been referred to in considering the diseases individually, but a partial résumé may be given here:

Leucocytosis is especially important as an evidence of inflammation, the increase being chiefly in the polymorphonuclear cells. It is well seen in pneumonia, cerebrospinal fever, and diphtheria. It is often present in scarlet fever, rheumatism, tonsillitis, variola, and erysipelas; but is usually absent in measles, rubella, mumps, typhoid fever, malaria, grippe, and tuberculosis except tuberculous meningitis. It is especially marked when inflammation is of a suppurative nature, provided the organism of the patient is showing a power of reaction, and hence is often observed in appendicitis, empyema, sepsis and many other conditions. A lymphocytosis is seen in pertussis and is characteristic of some forms of leukemia. An eosinophilia may be observed in leukemia, certain diseases of the skin, asthma and when intestinal parasites are present.

In determining the presence of a leucocytosis, and especially the relationship of the polymorphonuclear cells to the lymphocytes, the age of the child must always be considered, especially the fact that the lymphocytes are more numerous in infancy and early childhood than in adult life. (See Vol. I, p. 59; Vol. II, p. 455.) The discovery of leucocytosis in disease is of value both from a diagnostic and from a prognostic point of view. Diagnostically it confirms the presence or absence of certain 'disorders, or aids in determining whether an inflammation is serous or purulent, the increase of leucocytes, and particularly of the polymorphonuclear cells, being greater in the latter. Prognostically it indicates, the diagnosis having been previously determined, whether the organism of the individual [is showing a proper degree of resisting power to the disease.

POLYCYTHEMIA

The condition characterized by cyanosis with the presence of red blood-cells in numbers much above normal is seen in childhood in cases of congenital heart disease. (See p. 126.) A great increase of the red cells may also occur in conditions of concentration of the blood, as when in infancy there has been a rapid loss of fluid from the system, as a result dentation at the side. In the cell-body neutrophilic granules can sometimes be demonstrated. The transitional cells are sometimes hard to differentiate from the polymorphonuclear neutrophile, and many authorities prefer to class them with these. Transitionals, in the 1st year of life, form 7 to 10 per cent. of the white cells, after which they fall to about 2 to 4 per cent. Any large increase in the number of transitionals is usually indicative of the development of a rapid increase of polymorphonuclear neutrophiles.

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[1] Amer. Jour. Dis. Child., 1915, IX, 358.

Polymorphonuclear Neutrophiles (37–41, in plate).—These are a little larger than the average red blood-cell. The nucleus is irregular, and is made up of from 2 to 5 parts joined together by narrow threads of chromatin, although occasionally the nucleus is merely one horseshoe-shaped mass. This is the type that is not easily differentiated from the transitional cell. The cell-body stains faintly pink and contains small neutrophilic granules, coloring neither strongly acid nor strongly basic. The normal percentage of polymorphonuclear cells during infancy and childhood is about as follows: 1st year 30 per cent., 2d year 40 per cent., 6th year 45 per cent., 10th year 55 per cent., at puberty about 55 per cent. to 60 per cent.

Polymorphonuclear neutrophiles are increased in conditions causing an absolute leucocytosis, although the relative percentage may be increased without an absolute augmentation of the number of leucocytes.

Polymorphonuclear Eosinophiles (42–45, in plate).—These are on the average a little larger than the neutrophiles. The nucleus is like that of the polymorphonuclear neutrophile, except that it does not stain quite as deeply. The cell-body contains very large, coarse granules which stain strongly with the acid stains, as eosin (red). The granules are very apparent, fill the cell-body, and cannot, when properly stained be mistaken for any other type of granule. The eosinophiles vary under normal conditions from 0.5 per cent. to 8 per cent. of the leucocytes, the average being 2 per cent. to 4 per cent.

The commonest cause of eosinophilia in childhood is the presence of intestinal parasites, as hook-worm, oxyuris, ascaris, and tapeworm. Various cutaneous diseases, such as herpes zoster, eczema, pemphigus, and psoriasis may also cause eosinophilia. It is usually present in bronchial asthma, and often in infectious diseases, such as gonorrhea, tuberculosis, and scarlet fever. The increase may be from 10 per cent. in some forms of irritation to 75 per cent. or more in the cases harboring intestinal parasites.

Polymorphonuclear Basophiles (basophiles; "mast" cells) (46, 47, in plate).—These cells are slightly smaller than the polymorphonuclear neutrophile. The nucleus resembles that of this cell, but the body of the basophile contains fairly large irregular, round to oval, basic granules. Normally they form 0.5 per cent. or less of the leucocytes.

Basophilia is found in some cases of chronic cutaneous diseases, in bone-tumor, and in myelocytic leukemia.

Myelocytes (48–56, in plate).—Myelocytes are larger mononuclear cells, at least twice the diameter of normal red cells. The large pale nucleus is round or oval, occupies at least half of the cell, and is usually central or almost so. There are three types of myelocytes, the neutrophilic, the basophilic and the eosinophilic, the distinction being in the type of granules found in the cell-body. This distinction is the same as that which exists between the various polymorphonuclear cells. (See this.)

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[1] Amer. Jour. Dis. Child., 1915, IX, 358.

SYMPTOMATIC ANEMIA

(Secondary Anemia; Simple Anemia)

Etiology.—The number and variety of causes are large. They may for the most part be divided into a few groups:

1. *Hemorrhage.*—This may be from various places, visible or concealed, rapid or gradual. In the new born it may take place from the umbilicus or other wound, or internally, or it may occur in the course of the hemorrhagic disease with or without escape of blood from the body. In older infants and children it may be caused by certain intestinal parasites, or occur in typhoidal or other ulceration of the intestine and in many other conditions.

2. *Debility.*—Here are included a large number of causes. Infants may suffer from congenital asthenia and be anemic, or be so as the result of hereditary syphilis. Among other prominent causes of debility and anemia are rickets; insufficient or unsuitable nourishment; chronic digestive disturbances, and lack of fresh air. Still another class of cases depends upon the drain of long-continued albuminous discharge, as seen in nephritis or in chronic suppurative processes and chronic diarrhea. In older children there may be added to these causes the existence of chronic disease of the heart, or of the debility incident to rapid growth, confinement to bad air, and over-work in schools.

3. *Toxic and Infectious Causes.*—These may be organic or inorganic, developed within the body or entering it from without. The action of metallic poisons in causing anemia is inconsiderable in childhood. Much more important are the toxins produced in the course of various infectious diseases, especially diphtheria, malaria, and to some extent tuberculosis and syphilis; as well as those arising from decomposition of the intestinal contents or generated by intestinal parasites, or possibly by some of the internal glands, notably the spleen and seen in forms of hemolytic icterus.

Symptoms.—After a severe single hemorrhage there appear great pallor and syncope, and even death may result promptly. As a rule, however, the anemias of children are not due to this cause, and exhibit as the most marked symptom a gradually developing pallor of the mucous membrane and the skin. Although the degree of this indicates in a general way that of the anemia, it is often deceptive and in no wise to be depended upon. The amount of fatty tissue of the body is not necessarily diminished; but the muscles are flabby; fatigue comes on easily; there is often shortness of breath; the appetite is lessened, and gastro-intestinal disturbances are common. Anemic murmurs may frequently be heard over the base of the heart and in the vessels of the neck, and in well-marked cases a venous hum in the jugulars. The area of the cardiac dullness is often increased; enlargement of the spleen is not uncommon and the hands and feet are cold. Nervous symptoms are decided, especially in older children, consisting of fretfulness, irritability, headache and other neuralgic pains elsewhere, disturbed sleep, chilliness, and sometimes syncopal attacks. Infants with severe anemia may become very edematous.

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[1] Compt. rend. soc. de biol., 1892, XLIV, 384.

[2] Quart. Journ. of Med., 1908, II, 85.

The Blood.—The change in this is often rapid, depending upon the nature of the acting cause. The percentage of hemoglobin is reduced to 60 per cent. or 70 per cent. in moderate cases and to 20 per cent. or 30 per cent., or exceptionally less, in severe ones. In an instance of widespread subcutaneous hemorrhage in malignant diphtheria I obtained a record of 10 per cent. The number of red cells is also reduced, although to a less degree, 3,500,000 to 4,000,000 being seen in moderate cases, and even less than 2,000,000 in very severe ones. The low percentage of hemoglobin as compared with that of the red blood-corpuscles results in a diminution of the color-index, but not to the extent seen in chlorosis. The red blood-cells are paler than normal and in severe cases there is anisocytosis, poikilocytosis, and a few normoblasts, megaloblasts and megalocytes. The resistance of the corpuscles to hypotonic salt solutions is not constant, but is liable to be decreased (Hill).[1] The leucocytes may be moderately increased in number, but generally are unaffected, unless by the primary disease.

Course and Prognosis.—A single severe hemorrhage may prove immediately fatal. If not, there is usually a rapid restoration of the normal condition of the blood; but sometimes a more persistent anemia develops similar to that following other causes. The prognosis of secondary anemia of a chronic form depends in general upon the cause. The removal of this is necessary to establish a cure of the anemic condition. Few cases die directly as the result of the anemia, but oftener from some complicating disease, the power of resistance to which the condition of the blood has naturally diminished.

Diagnosis.—The diagnosis is based upon the blood-changes and the symptoms described, and the discovery of a cause to which the anemia is secondary. The differential diagnosis from other forms of anemia will be discussed when considering these.

Treatment.—Search for and removal of the cause is the first object sought. In this direction particular attention is to be given to the diet and hygiene, including the supply of abundant nourishing food, given frequently if the appetite is diminished; massage; the removal of nervous strain in school; the insisting upon life in the open air, and the like. Especially important in addition to this treatment in cases where the cause cannot be found or cannot be removed, are remedies to improve the condition of the blood. Here iron in some form heads the list. To be efficient it must be administered in sufficiently large doses. Among the preparations especially suitable for infants and small children are the pyrophosphate, lactate, citrate, and saccharated carbonate. A number of organic preparations of iron are on the market, some of them highly praised. It is questionable in my mind whether they equal in value those mentioned, and their iron-content is relatively less. To older children may well be given mass. ferri carb. (Vallet's mass), pil. ferri carb. (Blaud's pill) or ferrum redactum. In other cases arsenic is of avail, alone or in combination with iron. For this purpose the liquor potassii arsenitis may be used. In obstinate cases good results are often obtained through the administration of arsenic and iron hypodermically. The following formula is serviceable: Ferri citras viridis gr. 1 (0.065), sodii cacodylas gr. ½ (0.03), sodii glycerophosphas gr. 1½ (0.1), aqua to make m. 15 (0.9). This solution should be sealed in ampoules, and an injection of this amount given daily or every other day. Infants may receive from ½ to 1 grain (0.03 to 0.065) of the iron and ⅛ to 1/12 grain (0.008 to 0.005) of the cacodylate. Some cutaneous irritation and rise of temperature occasionally result, in which event the treatment may be omitted for a few days. Still other cases are especially aided by cod-liver oil; and in some instances I have had good results only after the administration of bone-marrow in addition to the treatment with iron.

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[1] Arch. Intern. Med., 1915, XVI, 809.

Improvement is sometimes rapid, but often slow; and the treatment may need to be long-continued without permitting of discouragement. Relapse is very likely to occur if treatment is stopped early after apparent cure.

Leishman's Anemia. *Infantile Kala-azar.*—In this connection brief reference may be made to a form of infectious secondary anemia seen in tropical countries and in the Mediterranean basin, and especially liable to attack children chiefly in the first 3 years of life. It is increasing rapidly in Italy, and Jemma[1] reported on 110 cases in children seen by him in Palermo during 5 years. The cause is the entrance into the body of a flagulated microörganism of the Leishmania group. This is found in enormous numbers in the liver and bone-marrow. The *symptoms* consist of an irregular fever of sudden onset; emaciation; cachexia; great enlargement of the spleen and later of the liver, and anemia. The skin is irregularly sallow and pigmented, and may exhibit icterus. There may be swelling and pain in the joints. The blood exhibits the changes seen in other forms of secondary anemia, but without increase of the total number of the leucocytes, and often a marked leucopenia, although the lymphocytes are relatively increased. The duration is several months or years, and the prognosis is very unfavorable, although recovery occasionally takes place. Antimony is recommended by Jemma[2] and Caronia,[3] and splenectomy by de Souza.[4]

CHLOROSIS

(Chlorotic Anemia)

This is generally regarded as a primary anemia. It is rarely observed under the age of puberty, and consequently an extensive discussion of it is not needed here.

Etiology.—The cause of chlorosis is not clearly understood. The disease seems sometimes to be hereditary and is much oftener seen in females, and after puberty practically only in this sex; but before this period males also may be occasionally attacked (Hayem).[5] Whether the cases occurring in children are true chlorosis is disputed; but at least they present the blood-picture which characterizes this disease. Cases in later childhood have been reported by Cantrel,[6] Förster[7] and others; and

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[1] Monatsschr. f. Kinderheilk., Orig., 1914, XII, 659.

[2] La Pediatria, 1916, XXIV, 1.

[3] Ibid., 65.

[4] Arch. de méd. des enf., 1915, XVIII, 349.

[5] Du sang, 1889, 745.

[6] Ref., Immermann, in Ziemssen's Handb. d. spec. Path. u. Therap., 1875, XIII, 1, 533.

[7] Gerhardt's Handb. d. Kinderkr., 1877, III, 1.

Leenhardt[1] and Biehler[2] described the blood-picture of chlorosis occurring not infrequently in infancy.

Among the many theories of the nature of the cause are that the affection depends upon a congenital hypoplasia of the arterial system; an auto-intoxication from chronic constipation; a disturbance of the production of lymph resulting in a watery condition of the blood; an interference with the internal secretion of the sexual organs; a defect in the absorption of iron. Among immediate causes are such agents as a lack of fresh air; insufficient or improper food, and emotional disturbances.

Symptoms.—As seen in typical cases in older subjects, these consist of fatigue; shortness of breath, palpitation, vertigo, and fainting coming on after exertion; digestive symptoms, especially constipation and cardialgia; diminution or perversion of appetite (pica), and frequently headache. The subcutaneous fat may be not at all diminished. The color is peculiar, being not only that of pallor, but with a yellowish-green tint. Edema of the ankles may occur. Anemic cardiac murmurs are audible over the heart and the large vessels in the neck, and a venous hum may be present. The heart may show evidences of dilatation, especially of the right side.

Blood.—The principle characteristics are a lowered specific gravity and a marked diminution in the percentage of hemoglobin, frequently to 30 per cent. or 40 per cent., while the number of red blood-corpuscles is little if at all reduced, although they are much paler than normal. The color-index is thus lower than in any other form of anemia. There is usually no alteration in the number of leucocytes. In severe cases there may be poikilocytosis and occasionally a few normoblasts.

Course and Prognosis.—The prognosis is usually excellent, thus indicating that certainly not all the cases can depend upon any congenital arterial hypoplasia. Recovery is most difficult in those in whom the disease began in childhood. Relapses are very prone to occur. The course is long, several months or possibly years being required before a permanent normal hemic state is reached. There is no fatality from the disease, except through the occurrence of some complication.

Diagnosis.—The symptoms in general, especially as regards cases developing in children, are uncharacteristic, being similar in most respects to those of severe secondary anemia; but the examination of the blood reveals the condition peculiar to the disease.

Treatment.—This is usually finally very satisfactory, the disorder yielding to the continued employment of iron in full doses. Appropriate remedies should be given for the relief of cardialgia and especially of constipation. The diet and hygiene need careful attention. Change of air is often very beneficial.

PERNICIOUS ANEMIA

(Crytogenetic Anemia)

By this title is designated an anemia, believed to be primary, of a most severe type and progressively increasing; often called idiopathic and exhibiting characteristic blood-changes. The first clear description was given of it by Addison[3] in 1855.

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[1] Thèse de Paris, 1906.

[2] Arch. de méd. des enf., 1913, XVI, 196.

[3] Constitutional and Local Effects of Disease of the Suprarenal Capsules.

Etiology.—The nature and causes are not certainly understood, and this is particularly true of childhood. It is very rarely seen in early life, especially in infancy, some of the reported cases having been atypical and probably symptomatic in nature. There is reason, however, to believe that others are genuine instances. Monti and Berggrün[1] collected 16 cases in children. Rotch and Ladd[2] report an instance in an infant of 9 months and refer to 6 other published cases in infancy. Cabot[3] estimated that a careful study showed less than 10 well-authenticated cases occurring in the 1st decade. At least 1 case has come under my own observation, in which the symptoms were typical. Among the many causes mentioned are a primary disease of the bone-marrow; a defective hemogenesis; the hemolytic action of some poison absorbed from the alimentary tract, as especially urged by Hunter;[4] syphilis; severe rickets; bothriocephalus latus and the anchylostoma; and atrophy of the gastric mucous membrane. It is disputed whether cases apparently dependent upon some of these causes are not to be excluded from the category of pernicious anemia and to be designated symptomatic, even though the clinical symptoms and the blood-picture are characteristic. Strictly speaking they should not be included here.

Pathological Anatomy.—There is found at autopsy an extreme pallor of all the internal organs, with capillary hemorrhages; fatty degeneration of the cardiac muscle and often of the kidneys and liver; and an unusual deposit of iron in the cells of the liver. There is evidence of a marked hemolysis taking place during life, and evidence, too, of an increased and perverted blood-formation. In a study made by Burr and myself[5] upon the livers of a large number of anemic conditions, there was never in any other disorder a deposit of iron comparable in any degree with that seen in pernicious anemia. It is present also in the spleen and kidneys to a limited extent. The marrow of the long bones is changed to red marrow, but this is not characteristic since we were able to produce it experimentally by repeated bleedings. In any event it is a normal condition in infants. There are very commonly degenerative changes in the posterior columns of the spinal cord.

Symptoms.—The symptoms develop slowly or sometimes rapidly and are those characteristic of any very severe anemia. There are prostration; debility; headache; gastro-intestinal disturbances, including vomiting, often diarrhea, and loss of appetite; faintness and dyspnea on exertion. The skin and mucous membrane are very pale, the former exhibiting a slightly yellow tint. There is no necessary emaciation. Edema is frequently present, especially about the ankles and feet, and there is often fever. Anemic murmurs are audible over the heart, the pulse is increased in rate, and palpitation and cardiac dilatation are observed. Hemorrhages from the skin and from the mucous membranes are of comparatively frequent occurrence. The urine is scanty and pale, or dark-colored from urobilin. There is sometimes in children a modrate enlargement of the spleen to be felt on palpation.

Blood.—The characteristic features are a watery condition with lowered specific gravity, diminished coagulability, and a great reduction of hemoglobin reaching 30 per cent. or lower. The percentage of erythrocytes is still more diminished, the number being sometimes as low as 1,000,000 or less. As a result of this condition the color-index remains at or about 1, or even is slightly higher. In no other form of anemia is this the case. The red blood-cells do not form rouleaux; their size is very variable, but they average larger than normal; and poikilocytes, megaloblasts, and normoblasts are present. Microcytes are often found in considerable numbers, and there is a decided preponderance of megalocytes. The presence of these altered blood-cells constitutes one of the chief characteristics of the disease. The percentage of leucocytes is diminished, especially of the polymorphonuclear cells, while the number of lymphocytes remains about normal, and a few myelocytes may be found. The resistance of the red cells to hypotonic salt solutions is increased.

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[1] Die chronische Anämie im Kindersalter, 1892, 102.
[2] Arch. of Pediat., 1901, Sept.
[3] Osler and McCrae, Modern Medicine, 1915, IV, 620.
[4] Lancet, 1888, II, 555.
[5] Transac. Assoc. Amer. Phys., 1891.

Course and Prognosis.—The course of the disease is usually progressively onward, with advancing weakness, apathy, and destruction of the blood. Exacerbations and temporary remissions occur, the symptoms improving during the latter, and the condition of the blood as well. The duration may be as much as a year, but is often only a few months. Not all cases, however, end fatally, and instances which appear to have been undoubtedly pernicious anemia have terminated in recovery.

Diagnosis.—This rests especially upon the extreme anemia; great reduction of red blood-cells and of the hemoglobin, yet with the high color-index of the blood; the presence of megaloblasts in considerable numbers; and the lack of increase of leucocytes. It is to be remembered that megaloblasts in small numbers may occur in other severe anemias, especially in young children. The increased resistance of the red cells to hypotonic salt solution is in marked contrast to the condition obtaining in the anemia of hemolytic icterus. It is to be noted that pernicious anemia, especially as it occurs in children, in some cases appears to be allied to other diseases of the blood, and capable seemingly of being transformed into them. In 1 instance under my observation the blood-picture was at first that of pernicious anemia, but the child later developed a typical and fatal leukemia.

Treatment.—Careful search should be made for a possible cause, such as intestinal parasites or alimentary intoxication, in the hope that the diagnosis may be a mistaken one. In addition the dietetic and hygienic measures recommended for symptomatic anemia should be followed. Of all remedies intended for a direct action upon the blood, arsenic offers the best hope. This should be given in gradually increasing amounts, until a fairly large dosage is reached. It may be combined with iron and given hypodermically as in secondary anemia. (See p. 460.) Splenectomy has been practised at times with apparent success; but appears as a rule able to do no more than produce a temporary improvement. It may, however, be undertaken in the hope of prolonging life.

APLASTIC ANEMIA

This is an uncommon form of anemia allied to pernicious anemia in many of its symptoms, but differing essentially in the fact that the bone-marrow fails to develop evidences of the regenerative hyperplasia of that disease. Whether the two conditions are but forms of the same malady has not been definitely determined. The disorder was first described by Ehrlich.[1] As seen in children it has been studied especially by Kleinschmidt,[1] who adds 4 cases personally observed to the 4 instances in children collected by Hirschfeld.[2] A case in an infant of 2 years is reported by Heubner.[3] The cause is entirely obscure. The characteristic lesion consists in a disappearance of the erythroblastic tissue of the marrow, which is either intensely yellow, or, if still presenting any reddish color, is shown by microscopical examination to be extremely poor in cellular elements. The **symptoms** are for the most part those of pernicious anemia, but with a much greater tendency to hemorrhage. Urobilin was not found in the urine of any of Kleinschmidt's cases. The course is rapid without remissions, and death occurs in a few months.

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[1] Charité Annalen, 1887, XIII, 300.

Blood.—The appearance of the blood is very characteristic. The erythrocytes are much diminished in number as in pernicious anemia, but, differing from this disease, there are few if any normoblasts or megaloblasts, and no poikilocytosis, anisocytosis or polychromatophilia. The color-index is usually low, but to this there are exceptions. The number of leucocytes is even lower than in pernicious anemia, with a marked relative increase of lymphocytes.

PSEUDOLEUKEMIC ANEMIA

(Splenic Anæmia. Anæmia Splenica Infantum. Anæmia Infantum Pseudoleucæmica)

The relationships of this not infrequent disorder have been the subject of much discussion. It has been regarded as a primary disease of the blood by some, and as secondary by others; as closely related to leukemia; or as representing in infancy the pernicious anemia of adults. The decided splenic enlargement has allied it to disorders of the spleen. That it exists at all as an independent disease is with much reason disputed. It is, however, at least a convenient clinical classification of cases much resembling each other, but which probably are atypical forms of different hemic disorders, chiefly pernicious anemia, secondary anemia, and leukemia. Especial attention was drawn to it by the writings of von Jaksch[4] and the title "anæmia infantum pseudoleucæmica" was applied to it by him.

Etiology.—The condition is seldom seen after the first 3 years of life, much the largest number of cases being observed in the first 2 years, oftenest beginning probably about the end of the 1st year. It is not often seen before the age of 6 months. The direct cause of the disease is unknown. It has been attributed to syphilis and to rickets, but without good reason, since the occurrence of pseudoleukemic anemia bears no relationship to the severity of these diseases, although it is true that rickets is a very common attendant. Sometimes there is a history of early digestive disturbances, improper feeding, or imperfect hygiene. That the enlargement of the spleen is the cause of the anemia seems unlikely. The disease has also been considered a final result of some toxic or infectious cause. Lehndorf[5] considered it only a myelogenous leukemia peculiar to infancy. Aschenheim and Benjamin[6] denominated it "rachitic splenomegaly."

Pathological Anatomy.—The striking feature is the great enlargement and hardness of the spleen, which shows no special change on microscopic examination. The liver is similarly somewhat enlarged and harder than normal, and there is sometimes moderate hypertrophy of the lymphatic glands of different regions. The condition of the bone-marrow varies, there being little change in most instances; in others a moderate increase of the cellular elements.

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[1] Jahrb. f. Kinderh., 1915, LXXXI, 1.

[2] Folia hematologica, 1911; Archiv, XII, 349.

[3] Folia hematologica, 1915; Archiv, XIX, 347

[4] Wien. klin. Wochenschr., 1889, II, 435; 456.

[5] Jahrb. f. Kinderh., 1904, LX, 194.

[6] Deut. Arch. f. klin. Med., 1909, XCVII, 529.

Symptoms.—The onset is slow and insidious. The degree of the anemia attained varies, but in well-marked cases the pallor is very decided, often with a slightly icteric tint. There is debility, fretfulness or apathy, and often the evidences of gastrointestinal disturbance and of rickets. There are also present the dyspnea and the cardiac symptoms witnessed in any form of anemia. The subcutaneous fat may be reduced or the children be plump. The abdomen is distended; the muscles are flabby; scattered small petechiæ are not infrequent, and there may be edema about the ankles. Fever is generally absent.

The most characteristic symptom is the decided increase of size of the abdomen, due principally to the enlargement of the spleen (Fig. 396). Palpation shows the spleen smooth, hard, sharp-edged and without tenderness. The notch can often be readily felt, and in the majority of cases the organ extends downward as far as the crest of the ileum, and transversely to the navel or beyond it. The hypertrophy may, however, be much less than this. The liver may sometimes be discovered slightly enlarged, reaching one or two finger-breadths below the costal border. Slight increase of size of the superficial lymphatic glands can be discovered by palpation in some cases.

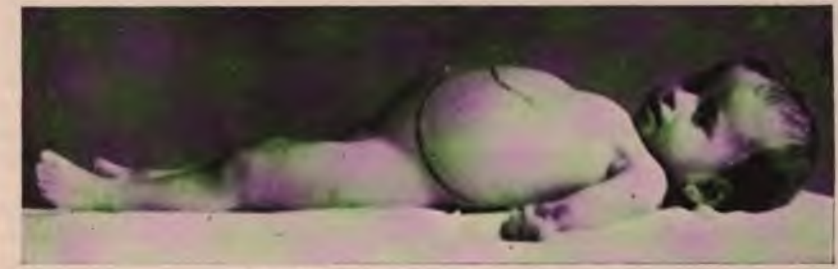

FIG. 396.—PSEUDO-LEUKEMIC ANEMIA.

Outline shows the lower boundaries of the spleen. From a patient in the Children's Ward of the Hospital of the University of Pennsylvania.

Blood.—Although there is some variation, there are still certain features believed to be characteristic. The specific gravity is reduced, and the formation of rouleaux may be absent. The percentage of hemoglobin is slightly diminished in mild cases, but much lowered in typical ones, reaching in severe cases 30 per cent. or less. That of the red blood-corpuscles is also reduced, in occasional instances to 1,000,000, but generally by no means to so great a degree, and the reduction does not keep pace with that of the hemoglobin, the color-index consequently being low. In well-marked cases the erythrocytes present a great variation in size and appearance, there being megalocytes, microcytes, poikilocytes, and polychromatophilia. Normoblasts are usually present in large numbers and megaloblasts may occur also, and may even exceed the percentage of normoblasts in some instances. Oftener than not there is an increase in the number of leucocytes, varying from 15,000 to 50,000 or higher; yet in many cases assigned to this category no increase has been found. The severity of the disease does not appear necessarily to be correlated with the leucocytosis, although the mild cases generally have fewer leucocytes than the severe. Probably the lymphocytes show a relative increase in the majority of instances, but this is inconstant and variable. The eosinophiles exhibit no constant variation; and mast cells may be found in small numbers. Myelocytes are nearly always present and sometimes in considerable quantity.

There is, therefore, nothing positively diagnostic in the blood-picture, although the usual combination of diminution of red blood-corpuscles, greater diminution of hemoglobin, leucocytosis, nucleated red blood-cells, and myelocytes is suggestive.

Course and Prognosis.—The course is slow, the symptoms lasting perhaps weeks in the milder cases, or months in others; meantime presenting little change, or a gradually increasing anemia and splenic tumor. Generally the final prognosis is good, the symptoms slowly disappearing, and complete recovery taking place. Yet, perhaps about 25 per cent. of the cases are not so fortunate, the patient often dying from a chronic bronchopneumonia, diarrhea, or other complicating disorder. In some reported instances the blood-picture has toward the end changed rapidly to that of leukemia. Whether these cases were in fact leukemia from the beginning, or whether this disorder occurred as a complication of a pseudoleukemic anemia cannot be definitely determined. In the forming of a prognosis it is to be remembered that even very unpromising cases with severe symptoms may yet recover completely.

Diagnosis.—This is, as a rule, not difficult. It rests chiefly upon the combination in infancy or early childhood of decided anemia and enlargement of the spleen. The blood-picture described is suggestive also, but not a positive diagnostic condition. Although some degree of splenic enlargement may accompany severe secondary anemia, or be dependent upon an accompanying rickets or syphilis, it by no means equals that of pseudoleukemic anemia. Myelocytic leukemia is distinguished by the much greater proportion of leucocytes and of myelocytes; lymphocytic leukemia by the great excess of lymphocytes; pernicious anemia by the hypoleucocytosis and the absence of very decided enlargement of the spleen. Confusion may, however, arise between the early stages of leukemia and those instances of pseudoleukemic anemia in which the leucocytosis chances to be decided; but the progress of the case will clear the diagnosis; in addition to the fact that leukemia of any form is very uncommon at this period of life. (See Leukemia, p. 467.)

Treatment.—With our lack of knowledge of the exact nature of the disease, our treatment must be empirical. A search must be made among the causes which have been enumerated as considered possible, and treatment instituted accordingly, with especial reference to alteration of the diet and the hygienic conditions. These and other measures to improve the general health are required, as described under Secondary Anemia. (See p. 459.) In the line of direct treatment the combination of iron and arsenic may be tried by the mouth or hypodermically (p. 460).

SPLENIC ANEMIA OF OLDER CHILDREN.—This is a condition, not infrequently observed, which is analogous in some respects to the pseudoleukemic anemia of earlier life. Inasmuch as the splenic enlargement appears here to be the most prominent factor, the subject will be found considered in the section upon Diseases of the Spleen.

LEUKEMIA

(Leucocythemia)

This affection was described independently by Bennett[1] and by Virchow[2] in 1845. The name indicates the light color of the blood. The two principal varieties, myelocytic and lymphocytic, were separated by the studies of Ehrlich and his pupils.[3]

Etiology and Pathology.—The disease in any form is comparatively uncommon in early life. Benjamin and Sluka[4] in 1907 were able to collect about 100 cases. It may occur even in infancy, and a leukemic condition of the blood in the new born has been reported, among others by Siefart[5] and Pollmann.[6] Adler[7] has collected in all 4 instances occurring at this period. If we separate the two classes, it will be observed that the myelocytic form is uncommon in early life and the lymphocytic form much more frequent than it. Pisek[8] in reporting a case stated that a careful search through medical literature showed only 19 authenticated instances of myelocytic leukemia in early life, of which 7 were less than 2 years of age. Knox[9] details an instance of myelocytic leukemia in an infant of 9 months, the youngest which he could find recorded. Adler[10] in reporting a case in a child of 5½ months could collect but 17 recorded instances of leukemia of any form occurring in the 1st year of life. Heredity has seemed to exert an influence in some cases. Males are oftener attacked than females. The direct cause and nature are unknown, and although the disease has been seen following simple anemia, pseudoleukemic anemia, syphilis, rickets, malaria, and inflammation of the throat, any direct relationship with these is undetermined. It has been claimed to be dependent upon a sporozoon (Löwit);[11] to be due to bacterial infection (Steele;[12] Simon and Judd[13]); to be a neoplasm of the bone-marrow; and to be of the nature of an auto-intoxication. Each of these views is in need of further confirmation. A review of the subject by Ward[14] led to the conclusion that there was very little positive evidence in favor of the infectious character of leukemia, and no instance of the inheritance of the disease. Whatever the cause, it results in a disorder of the hemapoietic system, producing a great modification of the leucocytes of the blood. According to the views of the Ehrlich School,[15] in one form of the disease the bone-marrow is affected, and in another the tissue of the lymphatic glands; but there is reason to believe that the bone-marrow is involved in every instance, and that in fact the two main forms are but varieties of one general disorder which affects all the blood-producing tissues in the body, the changes predominating in one region in some instances, and in a different one in others, and mixed forms occurring.

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[1] "Leucocythemia," Edinburgh Med. Journ., 1845, Oct. Ref., Griffith in Keating's Cyclop. Dis. of Child., 1890, III, 783.

[2] Froriep's Notizen, 1845, XXXVI, 151.

[3] Farbenanalytischen Untersuchungen zur Histologie und Klinik des Blutes. Gesammelte Mittheilungen, 1891, I.

[4] Jahrb. f. Kinderh., 1907, LXV; Ergänzungsheft, 253.

[5] Monatsschr. f. Geburtsh. und Gyn., 1898, VIII, 215.

[6] Münch. med. Wochenschr., 1898, XLV, 44.

[7] Jahrb. f. Kinderh., 1914, LXXX, 290.

[8] Arch. of Ped., 1916, XXXIII, 938.

[9] Amer. Journ. Dis. Child., 1916, XI, 462.

[10] *Loc. cit.*

[11] Verhandl. XVII Cong. f. inn. Med., 1899, 135.

[12] Boston Med. and Surg. Journ., 1914, CLXX, 123.

[13] Journ. Amer. Med. Assoc., 1915, LXIV, 1630.

[14] Brit. Journ. Child. Dis., 1917, XIV, 10.

[15] *Loc. cit.*

Pathological Anatomy.—The spleen is always somewhat enlarged, often filling the greater portion of the abdominal cavity. At first it is dark-red, soft and very hyperemic; later hard, with thickened capsule and perhaps adhesions. On section it is reddish-brown, firm, with areas of hemorrhage and others of grey-white, newly formed tissue. Microscopical examination reveals fibrosis and inflammation, with infiltration by lymphocytes, or in the myelocytic form by myelocytes. The liver is always larger than normal, and often much so; this being dependent upon an infiltration with cells, sometimes in distinctly localized areas. The lymph-glands of the body, including those of the mucous membrane of the alimentary tract, are much enlarged in some cases, little or not at all in others. They are infiltrated usually by lymphoid, but sometimes by myeloid, cells. The bone-marrow is always more or less involved. It is deep-red or dark-brown in color, or in some cases appears yellowish-green and puriform; or may exhibit hemorrhagic infarction; and on microscopical examination numerous nucleated erythrocytes, eosinophiles, myelocytes and polymorphonuclear leucocytes. There is a decided general disposition to hemorrhage seen, as in the spleen, bone-marrow, and in the form of petechiæ in the serous or mucous membranes. Some degree of lymphoid infiltration may be found in the heart-muscle, kidneys, lungs, thymus gland and even in the retina.

Classification.—Leukemia was formerly divided clinically into splenic, medullary, and lymphatic varieties, according as these tissues were chiefly or primarily involved. Later the first two were combined into splenomedullary, or splenomyelogenous, on the ground that the spleen bore no active causative part. It has been found, as stated, that the forms shade into each other, and that the cases shown to be lymphocytic by the examination of the blood may occur when there is enlargement of the lymphatic glands, when the lesions are largely limited to the bone-marrow, or when the spleen is greatly enlarged; and, on the other hand, that cases in which the myelocytes are a predominating element in the blood may exhibit no great enlargement of the spleen. It is true that, as a rule, cases with a marked development of myelocytes show great enlargement of the spleen, while a large excess of lymphocytes is attended by decided lesions of the lymphatic glands; but the exceptions which occur make it better to base a classification on the state of the blood rather than on the tissues affected. Such a classification would be into (*a*) myelocytic leucemia, acute and chronic (myelemia; splenomedullary, splenomyelogenous, or myeloid leukemia); (*b*) lymphocytic leukemia (lymphatic or lymphoid leukemia; lymphemia), and (*c*) atypical forms.

Symptoms.—These depend to a certain extent upon the type of the disease present. Several types may be considered:

1. ACUTE LYMPHOCYTIC LEUKEMIA.—This is the most frequent form in early life, 41 of Benjamin and Sluka's[1] 55 critically analyzed cases of leukemia being of this nature. It may develop insidiously with general malaise, pallor, loss of appetite, fever, and pain in the back or neck; but more often quite acutely, and the worst cases become seriously ill with entire abruptness. Not infrequently the first suggestive symptom observed, preceded for a few weeks by the vague manifestations mentioned, or without these, is hemorrhage into the skin or from some of the mucous membranes. This may be attended by moderate enlargement of the lymphatic glands, especially in the cervical region, irregular fever,

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[1] *Loc. cit.*

debility, and increasing anemia. The glands are not red or tender, and great enlargement of them is very unusual. Pain in the joints may occur, and diarrhea may develop. The spleen and liver become enlarged in nearly all cases, but only as a rule to a moderate degree. As the disease advances the hemorrhage from the gums, nose, mouth, or kidneys, and into the skin persists and may be severe; anemia and debility increase; fever continues; the glands usually grow slowly in size, but never attain the dimensions sometimes seen in chronic cases in adults, and edema may occur. In some cases leukemic infiltration of the skin has been observed; and papular, bullous, or other eruptions are not uncommon. In the very rapid cases the mucous membrane of the mouth and throat is swollen and often ulcerated; the tongue heavily coated; the breath foul; prostration is extreme; edema and dyspnea develop and the suddenness of onset and rapid course and severity of symptoms strongly resemble the result of some infection.

The *blood* is peculiarly pale and its coagulability is much diminished. The number of red blood-cells is much reduced, perhaps equalling 1,000,000 or less to the c.mm. in the severe cases, although the average is about 2,000,000. The hemoglobin ranges from 20 per cent. to 30 per cent. Normoblasts and megaloblasts may be present, sometimes in considerable numbers. Decided poikilocytosis is uncommon. The white cells may be increased from 50,000 or fewer up to a much larger number, but generally not more than from 200,000 to 350,000. Exceptionally the number is still higher, as in the case of an infant reported by Veeder,[1] giving 1,330,000 white cells per c.mm. The increase of leucocytes is in the lymphocytes, these constituting from 70 per cent. to even 90 per cent. of the total number of white cells. As a natural result the relative percentage of all other white cells is much diminished. The augmented percentage is, as a rule, chiefly in the larger lymphocytes. There is no increase in the eosinophiles or basophiles, and myelocytes are absent or are found in small numbers only. There occurs a great variation in the number of lymphocytes from time to time. Sometimes there is a sudden drop in the number found. This is especially liable to happen if any acute infectious disease develops, but it may be observed without this; or the number may be lowered, or oftener increased, toward the end of the disease.

2. Acute Myelocytic Leukemia.—This is an uncommon affection at any period, and in early life is rare. Benjamin and Sluka[2] found only 7 instances reported in their series of 55 collected cases of leukemia in infants and children. The onset is variable; sometimes with increasing debility and similar symptoms; sometimes with hemorrhage as in the lymphocytic variety. Ulceration of the mouth and throat has been reported. Fever is present; the spleen may be enlarged, but not to the extent seen in the chronic cases, and sometimes no enlargement is discoverable. The increase of the white cells is moderate, but always there is a characteristic excess in the percentage of the myelocytes, equalling from 10 per cent. to 50 per cent. of the total number of white cells found. Eosinophiles and mast cells may be absent. The number of lymphocytes is diminished.

3. Chronic Myelocytic Leukemia.—This is the type much oftenest seen, if we group all ages together; but in children it is comparatively

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[1] Arch. of Pediat., 1911, XXVIII, 43.

[2] *Loc. cit.*

uncommon. Thursfield[1] enumerates but 13 cases, including 3 of his own. The incidence is, however, certainly greater than this, and I can recall at least 3 cases in my own experience. The disease is insidious, perhaps with moderate debility and anemia; but often the enlargement of the abdomen is the first symptom attracting attention, and examination shows that this is dependent upon the spleen. As the disease advances the organ grows in size, until it may fill all the left side of the abdominal cavity, disappearing below the ilium and in the loin, and extending to the right beyond the vertical middle line (Fig. 397). It is hard, smooth, not tender, and reveals the characteristic notch. There is sometimes pain in the splenic region or in the bones. The lymphatic glands generally become enlarged to a moderate degree. The liver is slightly enlarged, or it may reach the level of the umbilicus. Fever is absent or occurs irregularly. Anemia becomes decided, although less than in the lymphocytic cases. Hemorrhage from any region is usually not an early symptom, but may develop later. Dyspnea and edema are among the later symptoms.

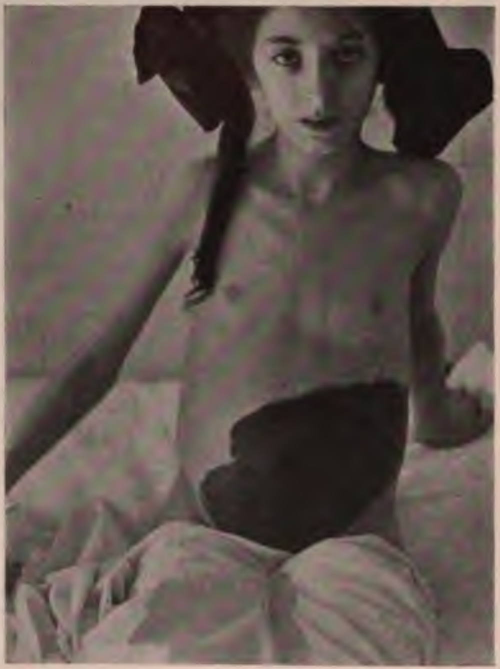

FIG. 397.—CHRONIC MYELOCYTIC LEUKEMIA.

Girl of 13 years, first seen June 1, 1915. Had suffered from loss of health and weight for 5 months. Spleen filled the entire left side of the abdomen, leucocytes 190,400, with 56 per cent. polymorphonuclears and 21 per cent. said to be myelocytes. Daily *x*-ray treatment instituted; Feb., 1916, leucocytes 14,000, and spleen could not be palpated with certainty, general health excellent. Later relapsed in spite of renewed *x*-ray treatment, failed in health, and died in the autumn of 1917. Black area shows position and size of spleen soon after treatment was commenced.

[1] Garrod, Batten and Thursfield, Diseases of Children, 1913, 519.

The **blood** is pale and coagulates slowly as in the lymphocytic form. The erythrocytes are much diminished in number, but rarely below 2,000,000 to the c.mm., and the hemoglobin is reduced proportionately. Normoblasts are present in considerable numbers, megaloblasts may be found, and polychromatophilia and granular degeneration are observed; but poikilocytes, microcytes and megalocytes are less common. The white blood corpuscles are usually enormously increased in number, reaching 200,000, or often much more per c.mm. The increase is seen in all the varieties, but the most important change is the presence of a large proportion of myelocytes, which constitute from 30 per cent. to 50 per cent. or more of the white cells. The increased percentage of lymphocytes is chiefly in the larger form. A characteristic peculiarity is the presence of the basophilic cells (mast cells) in fairly large quantity. The eosinophiles are above the normal in number, and some of them are mononuclear.

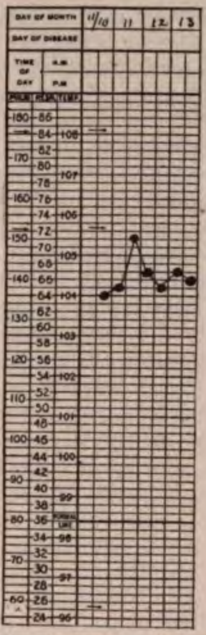

FIG. 398.—ATYPICAL LYMPHOCYTIC LEUKEMIA.

Marie B., aged 4 years. Admitted to the Children's Ward of the University Hospital, Nov. 10. Vague previous history. Temporary enlargement of glands in the neck in July accompanying sore throat; fever developed in August, followed by pain in the limbs and enlargement of the cervical and axillary glands, this enlargement having continued since that time. 2 weeks previous to admission pain in the limbs and abdomen, high fever, sweating, anorexia and rapid deterioration of general health. *Examination.*—Anemic, looks very ill, emaciated, bleeding gums, tonsils enlarged, enlargement of cervical and axillary glands and of the superficial lymphatic glands of the front of the thorax, spleen much enlarged, continued fever. *Blood.*—Hemoglobin 31 per cent., red blood corpuscles 2,620,000, leucocytes 3900. Differential count Nov. 13 gave lymphocytes 88 per cent., polymorphonuclears 5 per cent. Death Nov. 14. Autopsy showed extensive lymphocytic infiltration of various internal organs.

4. CHRONIC LYMPHOCYTIC LEUKEMIA.—This variety apparently does not exist in early life, in the sense of a long-continued disorder at all comparable to the chronic myelocytic form. With 2 exceptions, of 5 months and 7 months' duration respectively, none of the cases of lymphocytic leukemia in the series of Benjamin and Sluka lasted longer than 3 months.

5. ATYPICAL LEUKEMIA.—In cases of this nature there exist various divergences from the blood-pictures described. A certain number of them have been reported as "mixed" forms; dependent upon an unusually large number of lymphocytes in cases in general of a myelocytic type; or of more myelocytes than are ordinarily seen in lymphocytic cases. These variations are most liable to occur in children. In other instances of the lymphocytic type the total percentage of white cells may not be increased at all above normal; or even may be much below it; and it is only by the relatively large percentage of lymphocytes, in combination with other symptoms, that the diagnosis can be made (Fig. 398). In a case in a girl of 4 years with the diagnosis confirmed by autopsy the total number of leucocytes equalled only 3900 per c.mm., 88 per cent. of these being lymphocytes. Similarly cases of acute myelo-

cytic leukemia have exhibited a leucopenia, yet sometimes with a great preponderance of myelocytes. In the category of atypical cases is placed by some writers the *chloroma* previously mentioned. In this disorder there is present the blood-picture of leukemia with a tumor-formation in some locality arising from the bones. (See p. 453.) So, too, the condition already described as pseudoleukemic anemia (p. 464) is considered by Lehndorf[1] and others to be merely an infantile form of leukemia. The term *leukanemia* has been applied to the state in which the blood-picture and symptoms in general of leukemia are combined with those of pernicious anemia. While some of the cases probably belong to the latter disease, more are probably atypical instances of leukemia, in which there exists an accompanying great reduction of the red corpuscles with a high color-index. The white cells are generally lymphocytic.

Course and Prognosis.—The duration of acute lymphocytic leukemia is rarely longer than 6 or 8 weeks; sometimes extending to 3 months. Death is due to exhaustion, anemia from repeated hemorrhage, or some complicating disorder, such as diarrhea, nephritis, tuberculosis, or especially bronchopneumonia. Acute myelocytic leukemia likewise runs a rapid course to a fatal ending. Chronic myelocytic leukemia is slower in onset and more chronic in nature; but its duration is much shorter than in adult life, equalling 6 to 8 months, or occasionally 1 to 2 years. Death takes place from exhaustion. In the chronic forms of leukemia in adult life distinct spontaneous remissions often occur, accompanied by improvement in the general condition and in that of the blood. The more rapid course of the disease in childhood renders this less likely to take place. A temporary diminution in the number of white cells may, however, be occasioned by sepsis or some acute infectious disease, or be the result of treatment. The general severity and the duration of leukemia does not correspond in any direct way with the state of the blood.

The prognosis of leukemia is very unfavorable, although recoveries have been reported. It is probable, however, that most of these have been remissions occurring under treatment. One instance of myelocytic leukemia under my observation in a girl of 13 years might have been reported as cured by *x*-ray treatment, had not a further delay been made, during which the symptoms returned in force (Fig. 397).

Diagnosis.—This rests upon the character of the blood; the enlargement of the spleen or lymphatic glands and the general symptoms described, particularly the tendency to hemorrhage. The mere presence of a high polynuclear leucocytosis is in no sense a proof of the existence of leukemia. The disease is especially to be distinguished in children from secondary anemia with leucocytosis, and from pseudoleukemic anemia. Only a differential blood-count permits of this. A great increase of lymphocytes is characteristic of lymphocytic leukemia. The presence of mast cells and of a high percentage of myelocytes marks the myelocytic type. The blood of secondary anemia with leucocytosis often presents in children a relative multiplication in the number of lymphocytes, but never to the degree seen in leukemia; while in pseudoleukemic anemia, although the number of leucocytes is augmented, and myelocytes are present, there is no special increase of the lymphocytes, the total quantity of white cells is much less than in leukemia, and the myelocytes and mast cells are few as compared with myelocytic leukemia. In both forms of anemia mentioned the percentage of red blood-corpuscles is

[1] Jahrb. f. Kinderh., 1904, LX, 194.

usually lower than in leukemia. Very occasionally morbid growths of a sarcomatous nature situated in the thymus gland or in lymphoid tissue elsewhere produce a condition of the blood like that of leukemia. The diagnosis, however, is made easy by considering the other symptoms. Pertussis may show a large proportion or actual increase of lymphocytes, but can seldom cause any diagnostic difficulty. The forms of atypical leukemia described may present such difficulties in diagnosis that only time can solve the question.

Treatment.—This is extremely unsatisfactory. The acute cases will probably rapidly advance to a fatal ending no matter what therapeutic method is followed. The general and hygienic plan of treatment should be the same as for anemia of any sort. Arsenic in full doses is the best drug at our disposal, and under its use the number of leucocytes may undoubtedly be diminished. Benzol will without question reduce the number of leucocytes in some instances of myelocytic leukemia of the chronic form, but the action is only temporary. The dose for adults is from 3 to 5 grams (46 to 77 grains) daily, reduced proportionately to the age of the patient. It is a drug not without danger, and its employment should not be continued too long. The employment of the *x*-ray should certainly be tried. Not only the spleen but the long bones should be exposed. There is no doubt but that great improvement may sometimes follow; but that the treatment can effect permanent cure is uncertain. Extirpation of the leukemic spleen is not to be recommended.

DISEASES OF THE HEMORRHAGIC DIATHESIS

Here are grouped a number of morbid conditions, some of which are not clearly affiliated, while others shade into each other to a certain extent and may also bear a close relationship to other affections. The hemorrhagic disorders of the new born, such as melena, hemoglobinemia, and the like, have already been considered (Vol. I, p. 263), and the hemorrhagic tendency present in infantile scurvy has also been described (Vol. I, p. 604). None of these conditions are referred to here. There is often an intimate connection between forms of erythema and of purpura; while in nearly all of the infectious diseases a hemorrhagic tendency may at times develop, shown both by purpuric eruption and by hemorrhage from the mucous membranes or into the internal organs; and the same is true of sepsis, leukemia, pernicous anemia, aplastic anemia, some forms of icterus, and greatly debilitated states produced in any way.

In general in the hemorrhagic diathesis there exists either a congenital or a temporarily acting predisposing cause, in the latter case secondary to some other affection or appearing to be primary. There is a tendency to some change in the blood and blood-vessels which permits of the passing of the former through the vessel-walls. The direct causes are entirely unknown, and pathological and experimental studies have not been able to show just what the action of toxins or bacteria may be. Probably a variety of causes may produce identical symptoms.

The condition is conveniently divided into: (1) the *congenital form*, represented by hemophilia, and (2) the *acquired*, including the different varieties of purpura.

HEMOPHILIA

By this term is designated an inherited constitutional tendency to repeated severe and even uncontrollable hemorrhage produced by slight trauma or occurring spontaneously, the patient being known as a "bleeder."

Etiology.—The influence of inheritance is very remarkable. The disease is passed on from generation to generation entirely through the females, although the males of the family are those affected, and the females seldom, if ever. Grandidier[1] gives the proportion of male to female subjects as 13 : 1, while Bulloch and Fildes[2] after a careful study of 900 papers upon the subject, concluded that females never exhibit the disease. In the celebrated instance of the Appleton-Swan family the disease appeared in 7 generations (Osler).[3] Isolated cases are reported, but are rare, and some probably are only apparent instances of this, and a more complete history would show the influence of inheritance. Generally more than one child of a family is attacked. In probably 33 per cent. of the cases hemophilia makes its first appearance in the first 2 years of life, and rarely after childhood is past. In 111 cases referred to by Grandidier,[4] in which the age of onset was known, the first symptoms appeared in the first 2 years of life in 72 per cent. The general health of the patient exerts no influence, and the disease possesses no relationship to scorbutus, purpura of any sort, or the hemorrhagic disease of the new born.

The direct cause and the nature of the malady are unknown. Among causes suggested are abnormal fragility of the walls of the small blood-vessels; a high blood-pressure with abnormally small vessels; a diminished coagulability of the blood; a destruction of blood by the action of some infectious process; and a chemical alteration in the walls of the blood-vessels. Of these the only factor which appears to have a decided influence, since always present, is the very great retardation in coagulation of the blood. According to the investigations of Howell,[5] Hurwitz and Lucas,[6] and others, the diminished coagulability depends upon a lessened amount of prothrombin, the antithrombin being present in normal amount. It would appear also that there is no deficiency of fibrinogen, since the clot when formed is of the usual consistency.

Pathological Anatomy.—There are no characteristic lesions found. Abnormal thinness of the blood-vessel walls has been observed in a few fatal instances, but not in others. Hemorrhage may sometimes be discovered in and around the joints, and sometimes a synovitis may develop. The most evident feature is the excessive pallor of the tissues, the natural result in those who have died from hemorrhage.

Symptoms.—As a rule the disease does not manifest itself before about the age of 2 years, although it has been seen even in the new born, as shown by the hemorrhage which has followed circumcision. The statistics regarding the percentage of cases occurring in the 1st year are, however, probably not entirely conclusive, since some of them undoubtedly include instances of the hemorrhagic disease of the new born, which has no relationship to hemophilia. The symptoms consist solely in

[1] Die Hämophilie, 1877, 118.

[2] Treasury of Human Inheritance, London, 1911, XIV, A, 169. Ref., Pratt in Osler and McCrae Modern Medicine, 1915, IV, 717.

[3] Pract. of Med., 1909, 748.

[4] *Loc. cit.*, 124.

[5] Arch. of Int. Med., 1914, XIII, 76.

[6] Arch. of Int. Med., 1916, XVII, 543.

hemorrhage; the patient being healthy in other respects, except for the anemic condition which may persist if attacks of bleeding are frequent. Hemorrhage may take place either spontaneously, or, much more frequently, as the result of trauma. This trauma may be very slight, such as an insignificant blow on the nose, the extraction of a tooth, the use of a tooth-brush, circumcision or a slight wound of any other nature, including bruises followed by free bleeding under the skin. It is rarely seen in the umbilical wound in the new born, and, in fact, new-born infants of hemophilic inheritance rarely exhibit the symptoms of the disease at this period of life. The spontaneous hemorrhages may take place in any region of the body. They may be preceded by malaise, giddiness, irritability, sweating, chilliness, dyspnea, headache and other vague symptoms; or may come on without warning. Spontaneous hemorrhage occurs most frequently from the nose or mouth, but it may come from any other mucous membrane; or may be subcutaneous, producing a large hematoma or ecchymoses; or, in unusual instances, take place from the surface of the skin or into the muscles or serous cavities. A very characteristic seat for hemorrhage, either [traumatic or spontaneous, is into one or more of the joints, most frequently the knee or elbow. There occurs more or less rapid effusion of blood here, accompanied by fever, swelling, pain, heat and redness; or in some cases showing little pain and no discoloration.

Blood.—The blood exhibits no positive characteristic changes, other than those naturally seen in symptomatic anemia, and the greatly delayed time of coagulation which is now generally admitted to be present. It is important, however, in making the test to obtain the blood from a vein, and thus avoid the influence of the tissue-juices. There is a slight diminution in the number and relative percentage of the polymorphonuclear leucocytes. The blood-platelets are normal in number.

Course and Prognosis.—The disease is essentially a chronic one; the isolated occurrence of a very severe or persistent hemorrhage not being sufficient to constitute hemophilia. In the spontaneous cases there is a decided tendency to periodicity. The duration of a single hemorrhage is uncertain and variable. A slow oozing may continue for days until the patient seems almost exsanguinated or even dies as the direct result. The danger to life is not often immediate from the size and rapidity of the bleeding, but from its persistence in spite of efforts to stop it, and the consequent amount lost. There is seldom a fatal issue following the first hemorrhage. In less severe attacks there is often a remarkably rapid restoration to the normal condition of health.

The prognosis is on the whole unfavorable. Von Etlinger[1] estimated that 87 per cent. of the cases finally die of the disease; and in the 177 deaths in the series of Grandidier[2] 56 per cent. died in the first 7 years of life. The earlier in life the affection begins the more unfavorable is the prognosis, and conversely, the longer life continues the greater the possibility of the hemorrhagic disposition disappearing. This sometimes entirely ceases after puberty, but as a rule this is not the case. In those in which life is prolonged, repeated attacks of hemorrhage into a joint may result in chronic arthritic changes.

Diagnosis.—The chief diagnostic feature is the familial history of repeated hemorrhages difficult of control. Without this history the diagnosis is uncertain, since other causes may produce an abnormal tendency to repeated hemorrhages. Hemorrhages of this sort are seen oftenest in the form of epistaxis or recurrent hematuria; or there may be recurrent purpura, perhaps associated with some affection of the joints. A long-persisting hemorrhage controlled with difficulty is also not necessarily hemophilia. It is encountered, for instance, in some forms of icterus. The hemorrhagic diseases of the new vorn are not at all related to hemophilia, occurring as they do at a time when the latter disorder does not, as a rule, manifest itself. It is a striking fact that subjects of hemorrhage in the early weeks of infancy generally do not exhibit symptoms of hemophilia later in life, and that umbilical hemorrhage in the new born is of very rare occurrence in hemophilic families. There are, however, exceptions, and hemophilia does sometimes appear in the new born, as shown by the family history and the later history of the patient. From purpura and hemorrhagic disorders of an allied nature hemophilia is further distinguished by the presence in the blood of a normal number of platelets and by the delay in the coagulation-time. It is an interesting observation that in hemophilia a needle-prick for the purpose of obtaining blood can be made with impunity, while in some conditions of blood-dyscrasia this is not the case, and bleeding will be arrested with great difficulty.

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[1] Jahrb. f. Kinderh., 1901, LIV, 24.

[2] *Loc. cit.*, 154.

It is evident, then, that the diagnosis of hemophilia must be made with caution, and it is of great importance to make it correctly, as, for instance, in cases where a hematoma might otherwise be regarded as an abscess and be incised with serious results, or an affected joint might be considered rheumatic.

Treatment.—**Prophylaxis** is very important. In families known to be bleeders, marriage of the females should be discouraged. Even in the case of a child with hemophilic antecedents who has as yet shown no symptoms, operations should be avoided as far as possible, and accidents which might produce hemorrhage must be guarded against. Vaccination, however, has not been found to be dangerous. Of drugs employed for internal administration as prophylactic measures, the greatest attention has been paid to calcium lactate, which is supposed to increase the coagulability of the blood. A daily dose of 15 grains (0.972) or more can be given during several days of each week. Little benefit can, however, be expected, since the blood of hemophilics does not appear to be deficient in calcium. Thymus extract, ovarian extract, and yeast have also been recommended. The actual value of any of these is questionable.

In the treatment of **actual hemorrhage,** the bleeding part should be kept elevated, and compresses, tampons, or the cautery used, according to the situation and the demands. Trial may also be made locally of suprarenal extract or its derivative, calcium chloride, gelatine, fresh thymus extract, or chloride of iron. The subcutaneous injection of epinephrine, pituitrin, peptone, and gelatine respectively have their adherents. The last is not without danger on account of the difficulty of obtaining thorough sterilization. The subcutaneous or intravenous injection of a foreign serum, from another human or from the rabbit or the horse, increases the coagulability of the blood, and is one of the best procedures. In emergency the ordinary antidiptheritic serum can be employed for this purpose. When the loss of blood has been great, transfusion should certainly be done. After recovery from an attack, tonic remedies are indicated as in any form of anemia

PURPURA

By this title is designated the occurrence of more or less scattered effusions of blood beneath or into the skin, from the mucous membranes, or into the internal organs. The various purpuric affections are very intimately associated with each other, and no sharp line of demarcation can be established. The condition may be a secondary or symptomatic affection, or may be primary in the sense that it seems to be independent of any other discoverable morbid state. A convenient classification is into (1) Symptomatic Purpura and (2) Idiopathic Purpura.

1. Symptomatic Purpura.—The hemorrhages in this form are usually situated only in the skin, but to this there are exceptions. The purpura is merely a secondary symptomatic manifestation, the primary disease present being the predominant one. It does not differ in appearance from that to be described under the idiopathic form. In some cases with greatly lowered vitality necrotic areas may develop in the skin. A considerable variety of causes may be operative. They may be classified, following the arrangement given by Osler[1] into (*a*) infectious; (*b*) toxic; (*c*) cachectic; (*d*) neurotic and (*e*) mechanical; although the distinctions are by no means sharp.

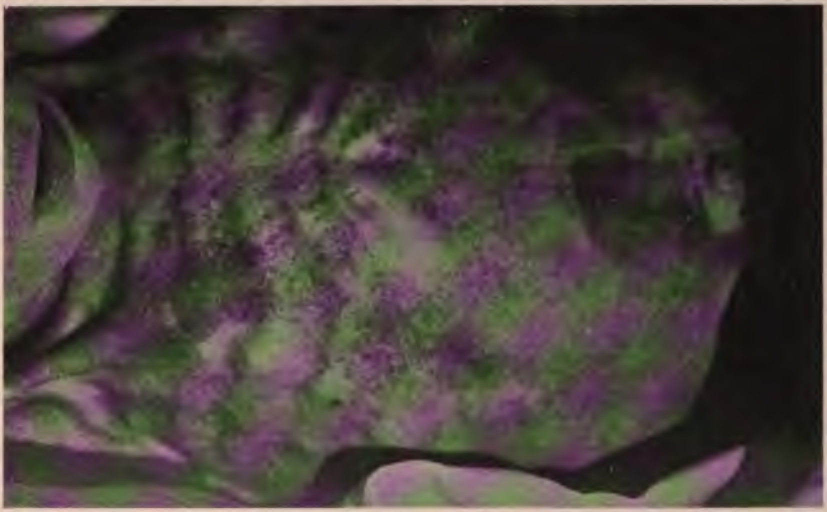

FIG. 399.—CACHECTIC PURPURA.

From a marantic infant of 10 months in the Children's Hospital of Philadelphia. Died a week after admission.

(*a*) **Infectious Purpura.**—This is a not infrequent form observed in septicemia, pyemia, malignant endocarditis and many of the eruptive and other infectious fevers, especially smallpox and cerebrospinal fever, and is a constant symptom of typhus fever. Cases have been reported without symptoms of meningitis in which meningococci were found in the blood from the hemorrhagic spots (Netter, Salanier and Wolfram).[2] Whether rheumatism may sometimes have purpura as a secondary manifestation, or whether "rheumatic" purpura is rather to be considered a primary disease of another nature (p. 481) is not definitely determined. Certainly, at least, typical cases of rheumatism very rarely exhibit any purpuric eruption or tendency to urticaria or erythema. In infectious purpura the spots may vary in size, from those of a petechial character to widespread subcutaneous and submucous hemorrhages.

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[1] Pract. of Med., 1917, 751.

[2] Brit. Journ. Child. Dis., 1917, XIV, 104.

(*b*) **Toxic Purpura.**—Poisons may be formed within the body which may produce purpura, as in cases of jaundice due to the bile entering the blood. Other substances introduced from without may have this effect, as, for instance, the venom imparted by snake-bites; or, in rare instances, the administration of certain drugs, among these being antipyrine, the iodides, copaiba, quinine, phosphorus, potassium chlorate, belladonna and diphtheria antitoxin. With the exception of the purpura of jaundice, the toxic variety is rare.

(*c*) **Cachectic Purpura.**—Here especially are to be noted the purpura occurring in greatly debilitated states in children and infants, as in the marantic condition produced by chronic diarrhea or other digestive disturbance (Fig. 399); or as often resulting from empyema, tuberculosis, prolonged bronchopneumonia and malignant growths. Here, too, is best placed the purpura seen in such diseases as leukemia, pernicious anemia, pseudoleukemic anemia, scurvy and Hodgkin's disease. In cachectic purpura the spots are usually small and occur chiefly on the abdomen and chest.

(*d*) **Neurotic Purpura.**—This form is very uncommon in early life. It may appear in hysteria, myelitis, locomotor ataxia and some other nervous disorders.

(*e*) **Mechanical Purpura.**—This may occur in pertussis from the violence of the cough; in epilepsy; when a limb is released from bandages; on the first use of the legs after a long illness, or whenever there has been venous stasis from any cause.

2. Idiopathic Purpura (*Morbus maculosus Werlhofii*).—The condition is not a very frequent one. The statement made of the lack of sharp distinction between the forms of purpura applies with especial force to the different divisions of the idiopathic or primary type. Once considered as distinct, they are now believed to be but variations in degree and localization of a single process. For convenience the division may be made into (*a*) purpura simplex; (*b*) purpura rheumatica; (*c*) purpura hemorrhagica; (*d*) Henoch's purpura, and (*e*) purpura fulminans; but these terms represent only types which shade into each other, and not in any way independent varieties. With the exception of the special symptoms characterizing each, and the prognosis, which varies with the type, all may be studied together.

Etiology and Pathogenesis.—Primary purpura is uncommon in infancy and oftenest seen in children after the age of 3 and up to that of 10 years. There is little difference in the frequency in the sexes. Among predisposing causes are debility, unhygienic conditions, digestive disorders, and anemia; but frequently purpura develops in children apparently in the best of health. The nature of any direct exciting cause is unknown. The disease strongly suggests on many occasions an infectious origin, and bacteria of various sorts have been found in the purpuric areas, or in the blood in some instances. Yet in most cases no such discovery has been made, and the variety of germs which have been described show that, if the disease is due to bacteria, as seems probable in many instances, certainly more than one form has the power of producing it. It is possible, too, that some chemical auto-intoxication may be operative, or that some alteration takes place in the blood-vessel walls, which permits of a passage through them of the red corpuscles. That the blood-vessels are affected in some manner appears certain, but whether this is an acute degenerative process, a bacterial embolism, or a vasomotor disturbance is not clear. It is possible that it differs with the case. The etiological relationship of changes in the blood to the eruption is uncertain.

Pathological Anatomy.—There is nothing characteristic found at autopsy. Local hemorrhages are discoverable into or under the skin or in the internal organs, or in the mucous membranes oftenest of the digestive tract. Ulceration or gangrene of the skin is very rarely observed, and the formation of blebs on the purpuric spots is also very uncommon. Bleeding into the joints does not occur. Degenerative changes have been found in the kidneys, and sometimes in the heart and liver in severe cases. Hemorrhages within the cranium or from the lungs are infrequent. In the bodies of those who have died of hemorrhage the usual anemic appearance of the tissues and organs is evident.

Symptoms.—The characteristic symptom of all types of purpura is the occurrence of hemorrhages into the skin. These may be of minute size (*petechiæ*) up to those of large area (*ecchymoses*). They are not elevated, their color is at first bright red but rapidly becomes a distinct purple tint, and they do not disappear on pressure. As they fade they assume a brownish and then a yellowish color. The spots often come out in recurrent crops. In the more severe forms of purpura hemorrhage also occurs from the mucous membrane of the nose, mouth, intestine, and urinary tract; and the subcutaneous hemorrhages are larger or more abundant. Other symptoms may attend or precede the hemorrhagic appearances, among them more or less fever, weakness, arthritic pain, digestive disturbances, and in some cases abdominal pain. The nature and degree of these symptoms varies with the type of the disease.

The Blood.—The changes are chiefly those associated with moderate anemia. There is a slight reduction of the specific gravity, the red blood-corpuscles, and especially the hemoglobin. The proportion of leucocytes is not materially altered except in the severe cases where a moderate leucocytosis occurs. A very characteristic alteration is seen in the blood-platelets in the severer cases (*purpura hemorrhagica*), these being much reduced in number, as shown by Duke[1] and others. Normally there should be about 450,000 platelets to the c.mm. (Pratt).[2] In purpura the number is frequently less than 100,000. Duke has also found that the "bleeding-time" is lengthened; *i.e.* after puncture of the skin with a needle the blood will continue to ooze into blotting paper, applied every 30 seconds, for longer than the normal time of from 1 to 3 minutes. A hemorrhagic areola is likely to form in the skin around the puncture. The coagulation-time is not altered, and no changes in the amounts of prothrombin or antithrombin were found by Howell.[3]

The special symptoms, course and prognosis may be studied more satisfactorily under the different clinical types.

(*a*) **Purpura Simplex.**—This is the mildest and most frequent form of idiopathic purpura. There may be slight indisposition for a few days, chiefly shown by loss of appetite, headache, diarrhea, vomiting and moderate fever; but often the attack begins suddenly, with the appearance of numerous, scattered, discrete, small, pin-head and split-pea sized, rounded, purple spots on some region of the body. The favorite sites are the extensor surfaces of the upper and especially the lower extremities; but the trunk may also be involved when the eruption is widespread (Fig. 400). The face and hands generally escape. The spots often come out in crops closely following each other, or sometimes in the form of recrudescences of the disease after intermissions. In some cases there is a distinct degree of edema in the affected region; or the individual purpuric spots may be edematous (*purpura urticans*).

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[1] Journ. Amer. Med. Assoc., 1910, LV, 1185.
[2] Osler and McCrae, System of Med., 1915, IV, 726.
[3] Arch. of Int. Med., 1914, XIII, 76.

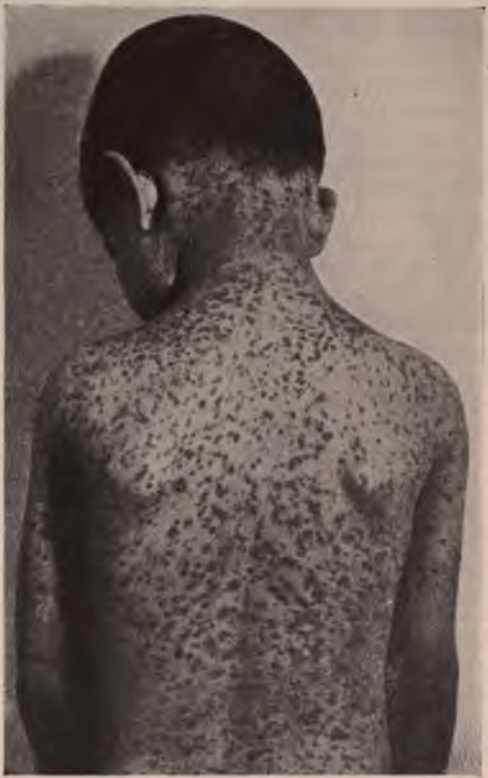

Fig. 400.—Purpura Simplex.
(*Hecker, Pfaundler and Schlossmann, Handb. d Kinderkr.*, 1906, *Ib*, 572.)

Sometimes urticaria and exudative erythema also develop, but this is less frequent than in rheumatic purpura.

Any prodromes observed soon disappear, and the *general symptoms* attending the eruption are absent or insignificant. There may be slight fever, but oftener none, or slight pain in the muscles and joints. The *duration* is from 1 to 3 weeks, or often longer, depending upon whether one or several crops of spots develop. Recurrences are very liable to take place some weeks or months after the attack is over. The *prognosis* is entirely favorable, except for the possibility of anemia after widespread eruption, the decided tendency for the disease to reappear later, and for the occasional passing of purpura simplex into a severer type.

(*b*) **Purpura Rheumatica** (*Peliosis rheumatica; Schönlein's Disease; Arthritic purpura*).—The characteristic of this type of purpura is the combination of the cutaneous eruption with pain and swelling of the joints, and often with urticaria and exudative erythema. The title "rheumatic" is misleading, as predicating an association with rheumatism, against the existence of which there is abundant evidence. The disease is less common in children than in young adults, and somewhat oftener seen in males than females. There are frequently decided *prodromal* symptoms; an exaggeration of those described under purpura simplex. In addition sore throat is a common early symptom, and there may be vague, not well-localized pains in the limbs. Then appears the purpuric eruption, similar to or often of smaller size than that in purpura simplex. Although it may occur in any part of the body, it is much oftener seen in the lower extremities, especially about the affected joints. It may appear alone, but is frequently combined with urticaria (*purpura urticans*), and in other cases with erythema multiforme, or, less often, erythema nodosum. Sometimes vesiculation of the purpuric spots occurs. At times the amount of edema is very decided, and this may involve the face and eyelids. Developing at about the same time with the eruption are pain and swelling in the joints, oftenest the knees or ankles, and usually multiple. The swelling is periarticular as well as from effusion into the joint itself. The latter is always serous; not bloody as in hemophilia.

The *general symptoms* are usually not severe. There is moderate or sometimes high fever, the urine may be scanty and may contain albumin, but without evidence of nephritis. Complications are not common. Endocarditis and pericarditis have been noted, but are very exceptional; another proof that the affection is in most cases not of rheumatic origin. The *duration* of the disease is variable, lasting 2 weeks and longer, depending, as in purpura simplex, on whether crops of eruption continue to appear with recrudescences of symptoms. The same tendency to repeated attacks is observed as in that type of the disease. The *prognosis* is favorable.

(*c*) **Purpura Hæmorrhagica.**—To this variety some writers especially apply the sub-title *morbus maculosus Werlhofii*; more using it as a synonym for idiopathic purpura of any sort. In this form there are not only the subcutaneous hemorrhages of purpura simplex, but from the beginning hemorrhage into and from the mucous membranes and into the internal organs. Vague prodromal symptoms of indisposition may last for some days, but oftenest the attack begins abruptly. The eruption is in part similar to that of purpura simplex; in part consists of ecchymoses of a size as large as, or larger than, the hand. It may be situated in any part of the body, including the face. In the most severe cases a large area of cutaneous surface, as perhaps an entire extremity, is edematous and purple-red from confluence of the hemorrhagic spots, and may occasionally exhibit vesiculation. The occurrence of erythema and urticaria is rare. The joints may be involved, but less often than in purpura rheumatica.

Cotemporaneously with the cutaneous eruption bleeding from the mucous membranes takes place. Hemorrhage from the nose is the most frequent, and hematuria is common, but bleeding may occur also from the intestine, or there may be hematemesis, or, less often, hemorrhage from the genitals, the ears, or the lungs. Ecchymoses may appear upon the visible mucous membranes, especially the palate, lips, tongue, and even the conjunctiva. The hemorrhage from the mucous membrane may be slight, or severe enough to cause death. The tendency to bleeding is so great that slight pressure upon, or scratching of, the skin may produce subcutaneous effusion or the free escape of blood from the surface; and a needle-prick may be followed by bleeding which is controlled with great difficulty.

The *general symptoms* vary with the severity of the attack. There may be irritability; prostration; headache; pallor, and continued moderate fever. The debility is such as to make the patient desire to be in bed, and the amount of blood lost in various ways may be so great that severe anemia results, and the patient may develop a condition of collapse with subnormal temperature. In other cases fever is absent or only occasional, and the subjective symptoms are little marked, except those which may be attributed to loss of blood. Some cases may pass into a typhoid state; or cerebral symptoms may be present, dependent upon intracranial hemorrhage or anemia. The spleen may be enlarged. Albuminuria may occur and edema, especially of the face and feet, may occur. Nehpritis may develop, but is less frequent than in Henoch's purpura. Vomiting is common. The *duration* of the disease in favorable instances is 2 weeks or longer. There is always great danger of recurrence, and this may take place again and again, prolonging the course for weeks or even months.

The characteristic of purpura hemorrhagica is the hemorrhage from the mucous membrane, and it may happen that this is the only symptom of the disease present, and that no cutaneous bleeding occurs at all. Such cases could be readily confounded with hemophilia, but are distinguished by the entirely different blood-picture.

The *prognosis* of purpura hæmorrhagica depends upon the amount of hemorrhage and the duration of the attack. Death may take place from exhaustion following loss of blood, or from intracranial hemorrhage. Sometimes the course is only a few days to a fatal termination. In general, however, the prognosis is good, and the majority of cases recover. Those developing the typhoid state nearly always die.

(*d*) **Purpura Abdominalis** (*Henoch's purpura*).—This type, to which special attention was called by Henoch[1] and which might be considered a variety of purpura hæmorrhagica, exhibits some of the features of this latter and others of the type of rheumatic purpura, with the addition of an unusual development of abdominal symptoms. It is observed chiefly in children and adolescents. It begins with the *symptoms* usual to rheumatic purpura, including the involvement of the joints, the urticaria and erythema, and the purpuric eruption, except that the last may early be slightly developed. Then, with an increase of these, severe abdominal manifestations suddenly appear, among them being very obstinate vomiting; paroxysmal attacks of intense abdominal pain; complete loss of appetite; constipation followed by diarrhea; tenesmus with bloody stools; not infrequently nephritis; enlargement of the spleen, and hemorrhage from the mucous membranes. The abdomen is hard and tender, especially in the region of the transverse colon, and the legs may be flexed upon it, the condition suggesting intestinal obstruction, especially intussusception. There is generally fever, and the patient rapidly becomes much debilitated. The single attack lasts only a few days, but is then followed in the course of a few days more, or perhaps a week or month, by another attack, and so on; the course being sometimes very long drawn out. Generally there are less than half a dozen attacks, the intensity growing gradually less, but Pratt[1] records an instance in which there were over 60 attacks at intervals of less than a month during 5 years. The predisposition to recurrence is especially marked in this type of purpura.

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[1] Berl. klin. Woch., 1874, XI, 664.

Not all the characteristic features are seen in each case, or even in each attack in the same individual. Arthritic involvement may be absent, and still oftener urticaria or erythema may fail to develop. The cause of the abdominal pain is not understood. It may be a thrombosis of the intestinal vessels, or effusion of blood into the mucous membrane of the stomach and intestines, or localized edema in these regions. The prognosis must be guarded in view of the prostration produced, the tendency to recurrence, and the danger of the development of a complicating nephritis or cerebral hemorrhage. Still, the larger majority of the cases in children recover. In 102 collected cases of all ages, as quoted by Pratt, 8 died.

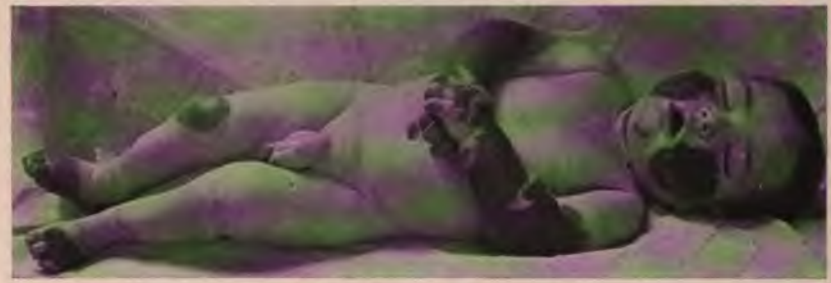

Fig. 401.—Purpura Fulminans.

Infant in the Children's Hospital of Philadelphia. Time and method of onset not clearly discoverable. Patient exhibited edema and large dark-purple effusions of blood. Mucous membranes apparently uninvolved. Autopsy negative.

(*e*) **Purpura Fulminans.**—This very unusual condition, to which the title was given by Henoch,[2] is seen oftenest in children, and is like purpura simplex in the limitation of the hemorrhage to the skin; but unlike it in the rapid development, and the extent and amount of the hemorrhage. The onset is very sudden, with chilliness, high fever, great prostration and vomiting. Ecchymoses of large size appear with remarkable rapidity, in the course of a few hours completely covering large areas chiefly on the extremities (Fig. 401). These are somewhat symmetrically placed, and of a uniform, dark-bluish or blackish-red color. The skin is hard, edematous, and may exhibit bullæ containing a bloody, serous fluid. The spleen is generally enlarged, and there is often albuminuria. The course is extremely rapid; suggesting a malignant infection or a toxemia, and death takes place in from 12 to 24 hours, or sometimes after 3 or 4 days, with cerebral symptoms ending in coma.

Diagnosis of Purpura.—The diagnosis of any form of purpura is usually easy, and rests in large part upon the occurrence of the characteristic extravasations of blood. Symptomatic purpura is recognized by its development after or in connection with some of the diseases mentioned as causative factors, while idiopathic purpura is distinguished by the absence of any such cause. Confusion might be possible between a purpura fulminans and some of the malignant, rapidly fatal, hemorrhagic secondary cases associated with the infectious fevers. These latter are characterized by the higher temperature, the prodromes, and the probable presence of some distinguishing symptoms of the causative disease. The nature of the hemorrhagic condition seen in lymphocytic leukemia and aplastic anemia is at once recognized by an examination of the blood. Scorbutus might suggest a primary purpura with articular involvement; but the onset in scurvy is slower, the history distinctive, the gums are spongy, and the painful regions are the shafts of the bones, not the articulations. The question whether "rheumatic" purpura is purpura with rheumatism has already been raised (p. 481). That many cases are certainly not rheumatic is indicated by the lesser degree of articular involvement; the lower temperature; the absence of sweating and of cardiac derangement, and the appearance of urticaria and erythema in conjunction with the purpura. Henoch's purpura presents difficulties in the cases where the purpuric eruption is not well-marked, and the abdominal symptoms very severe. Here the diagnosis of an intussusception, acute colitis, or gastric ulcer could readily be made; and this has, in fact, been done, and operations performed. On the other hand, a number of cases of intussusception occurring as a complication of Henoch's purpura have been reported (Morse and Stone).[1] A recurring purpura hæmorrhagica can be positively distinguished from hemophilia by the fact that in the latter the coagulation-time of the blood is greatly delayed, the bleeding-time is not prolonged, the blood-platelets are not diminished, and that simple puncture of the skin with a needle does not cause obstinate hemorrhage, unless a vein has been actually wounded. The reverse in all these particulars is true of purpura hæmorrhagica.

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[1] Osler and McCrae, Modern Med., 1915, IV, 709.

[2] Berl. klin. Woch., 1887, XXIV, 8.

The various types of primary purpura shade into each other, and together constitute one disease, a patient perhaps beginning with the symptoms of one type, and later developing those of another. The diagnosis between them is therefore a matter of interest only from the point of view of nomênclature. Purpura simplex is characterized by subcutaneous hemorrhages only; purpura rheumatica by the combination of these with urticaria and allied conditions, and with involvement of the joints; purpura hemorrhagica by the existence of hemorrhage from the mucous membranes, as well as in the skin; Henoch's purpura by the combination of the symptoms of rheumatic purpura with abdominal crises and hemorrhage from the mucous membranes; purpura fulminans by its rapid, fatal course, with the very extensive hemorrhage occurring only in the skin.

Treatment.—The treatment of symptomatic purpura is that of the primary disease. Measures should be taken to improve as rapidly as possible the impoverished condition of the blood, and to increase the general strength. Of all therapeutic measures for the idiopathic cases the most important is rest, recumbent in bed no matter how well the child feels; and this should be sufficiently prolonged; for it is a matter of frequent experience that a too early leaving of the bed is followed by a return of the purpura. The diet should be nourishing but easily digestible. On the ground that the disease might possibly be allied to scurvy, I have tried the administration of orange juice, but without noticeable effect. Nevertheless, a diet containing increased proportions of vegetables, fruits and similar foods, may well be employed. Plenty of fresh air is required. There is no known specific treatment for the disease. Various remedies have been advised, among them sulphuric acid, ergotin, arsenic, epinephrine, hydrastine, gallic acid and turpentine. Calcium lactate is also recommended, but there is no physiological reason why it can be expected to be of benefit, since the coagulability of the blood is not diminished in this disease. The treatment must, in the main, be symptomatic. Hemorrhage from the nose may demand local measures to control it, if severe; such as the application of a solution of epinephrine, or perhaps the plugging of the posterior nares. Severe bleeding from any region may be treated by epinephrine or gelatine internally, or the hypodermic administration of pituitary extract, epinephrine, gelatine thoroughly sterilized; or especially of normal human blood-serum or that of another animal. For the pain and vomiting of Henoch's purpura the application of an ice-bag may be tried, and, if necessary, a hypodermic injection of morphine and atropine given. During convalescence internal remedies are frequently needed to relieve the anemic condition of the blood. Change of climate to the sea or the mountains is often serviceable.

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[1] Arch. of Pediat., 1909, XXVI, 287.

CHAPTER II

DISEASES OF THE SPLEEN

ACUTE ENLARGEMENT OF THE SPLEEN

Enlargement of the spleen is very common in many either acute or chronic diseases, and this is particularly true of early life. An enlargement of moderate degree, lasting but a short time, is a frequent attendant upon most of the acute infectious diseases, being especially well marked in typhoid fever and malaria and common in acute tuberculosis and sepsis. At autopsy the spleen is found congested, deep-red and soft, and the Malpighian bodies may be increased in size. In sepsis and occasionally in typhoid fever infarcts may be present, and these may have advanced to the formation of abscess.

The **symptoms** usually consist simply in the discovery of the enlargement. With the child lying upon its back or on its right side, the physician's warmed finger-tips are held resting closely but lightly beneath the left costal border in the mid-axillary line. During inspiration the lungs force the spleen downward, and it can then be palpated if enlarged; although considerable practice is required if the increase is but slight. The abdominal walls should be relaxed, and the flexing of the child's thighs and the diverting of its attention may aid in accomplishing this. If the enlargement of the spleen is not at first discovered, the hand should be kept in position and the pressure gradually increased, the finger-tips pushing upward under the costal border. Inasmuch as even the normal spleen may sometimes be detected in this way in favorable cases, to constitute a moderate enlargement the organ should protrude an inch below the border of the ribs. Percussion of the spleen should also be made, but is unsatisfactory in infancy, and in no event can positive conclusions be drawn from it, unless confirmed by palpation. No treatment is required, and the enlargement disappears if recovery from the primary disease takes place.

Rupture of the Spleen.—This is a very rare sequel of acute enlargement in typhoid fever or malaria. The symptoms are those of hemorrhage into the peritoneum, and immediate operation is necessary.

CHRONIC ENLARGEMENT OF THE SPLEEN

This is dependent upon a variety of causes, and diverse pathological conditions result, some of which are discussed each under its individual title. The enlargement may be of moderate size or very great. All the *anemias* if severe and long-continued are liable to produce it, and leukemia, pseudoleukemic anemia and Hodgkin's disease may exhibit it unusually well developed. *Rickets* not infrequently is attended by a moderate and sometimes a very considerable increase of size of the spleen. The thinness of the abdominal walls and the defective development of the chest aid in producing the impression of enlargement in this disease by the allowing of a certain degree of downward displacement of the organ. In hereditary *syphilis* enlargement of the spleen of moderate degree is nearly always present among the early visceral manifestations of the disease; and is then discoverable at birth. This may disappear as age advances, but a very great enlargement of the spleen with anemia may occur as one of the symptoms of late hereditary syphilis as seen in later childhood. Miliary *tuberculosis* often results in a very decided enlargement of the spleen, depending upon the development of caseating tubercles. *Malaria* may produce a very great increase of size, and *hemolytic icterus* of different types is attended by splenic hypertrophy (see Hemolytic Icterus, Vol. I, p. 835; Vol. II, p. 489), and this is true likewise of *biliary cirrhosis.* The passive congestion from *cardiac disease* is another factor, and there is always enlargement, often very great, in *amyloid degeneration,* the principal causes of this change being syphilis, tuberculosis and long-continued suppuration. Finally increase in the size of the organ may rarely be the result of *morbid growths* in it. There are, as a rule, no symptoms attending secondary splenic enlargement other than those of the primary disease.

PRIMARY SPLENOMEGALY WITH ANEMIA

(Splenic Anemia; Banti's Disease; Gaucher's Disease; etc.)

This form of splenic enlargement has given rise to much discussion, and its causes and relationship are even yet far from being completely understood. The title "splenic anemia" was formerly employed to cover all cases of decided enlargement of the spleen accompanied by well-developed anemia. Increased knowledge of the blood and of the splenic functions has shown that the term comprises several distinct types, and on this account it would be better to abandon it. One of the forms, "pseudoleukemic anemia," has been described under Diseases of the Blood (p. 464), inasmuch as the anemia appears here to be the more important condition. Some of the varieties of splenomegaly are referred to in discussing Hemolytic Icterus (Vol. I, pp. 835, 836; Vol. II, p. 489). For many data concerning the types now under consideration I am indebted to the contributions of Krumbhaar[1] and of Lyon[2] as well as to those of other writers, to which reference will be made in course.

1. BANTI'S DISEASE

Etiology.—The not very common type of splenomegaly to which this title is now applied was first differentiated by Banti.[3] The cause is entirely unknown and there is no evidence of inheritance. The increased amount of pigment in the spleen and the recoveries which have followed splenectomy indicate that the principal elements producing the symptoms reside in this organ, as Banti maintained. The affection develops usually in young adult males and very seldom before later childhood.

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[1] Jour. Exper. Med., 1912–14, various references. Amer. Jour. Med. Sci., 1915, CL, 227.

[2] Osler and McCrae, Modern Med., 1915, IV, 954.

[3] Lo Sperimentale, 1894; Sez. Biol., XLVIII, 407.

Pathological Anatomy.—There is an increased amount of fibrous tissue in the spleen, and the Malpighian bodies are small and few in number, especially in the later stage of the disorder. Infarcts are sometimes found and an increased amount of pigment is usually present. Later in the course there is added an interlobular cirrhosis of the liver. The splenic vein, portal vein and other neighboring vessels are dilated and exhibit degenerative changes. There is no enlargement of the lymphatic glands.

Symptoms.—Splenic enlargement, anemia, and hepatic cirrhosis constitute the characteristics of the disease. The course is very slow and the symptoms may be divided into three stages. In the first, or pre-ascitic stage, lasting several years, there is increasing weakness and pallor; digestive disturbances and abdominal pain; a hard, smooth, progressively enlarging spleen; a tendency to hemorrhage, especially from the stomach; and moderate anemia of the chlorotic type. Very often, however, the anemia and hemorrhagic tendency do not develop until later. There is an excess of urobilin in the urine. The red blood-cells show no increase of fragility, and the number of white cells is slightly below normal. The second stage is of a few months' duration, and is characterized by scanty urine with an increase of urobilin; digestive disturbances and diarrhea; and slight augmentation of the size of the liver. The third stage is that of developing hepatic cirrhosis, with ascites, occasionally slight jaundice, bile in the urine, emaciation, anemia, and finally shrinking of the liver.

Prognosis.—Although the course is slow, lasting perhaps 10 years or more, the ultimate prognosis is unfavorable, unless operative treatment is employed. The patient may die before the stage of cirrhosis develops.

Diagnosis.—This is to be made largely by exclusion of other possible causes of splenomegaly. Early in the course the diagnosis may be impossible. Secondary anemia of any sort accompanied by splenic enlargement is recognized by the sequence of these conditions, the anemia being apparently the cause of the splenomegaly. Pseudoleukemic anemia exhibits a marked increase in the number of leucocytes, and develops at a much earlier age. Leukemia and pernicious anemia show characteristic blood-changes. Hemolytic icterus has discoloration of the skin as one of the earliest symptoms; often appears at an earlier period of life; and is positively distinguished by the increased fragility of the red blood-corpuscles. The urine contains urobilin, but no bile, and in the congenital type the general health is little affected. Gaucher's disease begins in infancy and childhood; its course is extremely slow; it exhibits often a familial occurrence; jaundice and ascites are absent; a brownish discoloration of the skin may occur; the spleen is generally larger, and the anemia less. Portal hepatic cirrhosis may resemble the later stages of Banti's disease very closely. However, the enlargement of the spleen in the former is the secondary affection; the course is shorter, and anemia is less marked. In Hanot's cirrhosis the liver is greatly enlarged and there is a chronic icterus. Syphilitic enlargement of the liver and spleen may also closely resemble Banti's disease; but there may often be discovered evidences of syphilis in the past history or the present condition of the patient.

Treatment.—The only treatment which has been of avail is splenectomy. The operation should, however, be performed early in the disease, before the liver becomes involved. It is in itself a serious one (27.4 per cent. for splenectomy for all causes, Johnston),[1] and we do not yet know how lasting may be the benefit which has been obtained. Giffin's[2] statistics indicate that it may be absolutely curative in many instances. Owing to the difficulty in distinguishing Banti's disease from syphilitic enlargement of the spleen, antisyphilitic treatment should always first be given a trial.

2. GAUCHER'S DISEASE

(Large-celled Splenomegaly)

This rare affection was first described by Gaucher[3] under the title of "primary epithelioma of the spleen." Brill and Mandelbaum[4] collected 14 cases which they considered could properly be included here, and Knox, Wahl and Schmeisser[5] add 2 other reported cases to the series, in addition to 2 new instances observed by them. Besides these Bernstein[6] records an instance in which the diagnosis was made by examination of the splenic pulp obtained during life; and possibly a few other reported cases might be included also. It is often distinctly familial, and most frequent in the female members in one generation. It is not hereditary. The principle **lesions** are now believed to consist in the deposit in the spleen of masses of very large, multinuclear cells of an endothelial character, which to a great extent replace the normal splenic tissue. They appear macroscopically as white spots or streaks. A similar change, although to a less degree, occurs in the liver, bone-marrow and lymphatic glands.

Symptoms.—The disease appears to begin insidiously in infancy and early childhood, although probably congenital, and runs a very chronic course lasting many years. Of the series of Knox and his collaborators 9 cases had not passed the age of 12 years when the abdominal enlargement was recognized, the youngest being but 2 months old at the time. The chief symptom is the progressive enlargement of the spleen, which is hard, smooth, regular in shape, and finally reaches an enormous size. The liver after a time enlarges also, although not to the same degree, and is of a greater size than in Banti's disease. The general health is in no way affected at first; but later anemia of the chlorotic type develops, which, however, is never very great. Leucopenia is a very characteristic symptom. Ascites and icterus are absent. There is often a brownish discoloration of the skin, especially of the face and extremities, and a peculiar yellowish wedge-shaped thickening of the conjunctiva on each side of the cornea. The superficial lymphatic glands may be very slightly enlarged. There may be submucous or subcutaneous hemorrhages in the early stages, but the gastric hemorrhage, so characteristic of Banti's disease, is absent. Further diagnostic differences from this affection have already been pointed out (p. 487). Death occurs from some intercurrent affection.

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[1] Arch. of Surgery, 1908, XLVIII, 50.

[2] Amer. Journ. Med. Sci., 1913, CXLV, 781.

[3] Thèse de Paris, 1882. Ref., Krumbhaar, Amer. Journ. Med. Sci., 1915, CL, 227.

[4] Amer. Journ. Med. Sci., 1913, CXLVI, 863.

[5] Johns Hopk. Hosp. Bull., 1916, XXVII, 1.

[6] Journ. Amer. Med. Assoc., 1915, LXIV, 1907.

In the line of treatment splenectomy has been tried. Hermann, Roth and Bernstein[1] record 9 reports of this operation and 6 operative recoveries. Temporary improvement after operation is reported, but it is too early to determine how permanent recovery may be. The fact that the affection is a systemic one, involving other tissues as well as the spleen, would make ultimate recovery after operation seem unlikely.

3. HEMOLYTIC ICTERUS

The forms of this affection have already been referred to in discussing icterus in the section upon Diseases of the Liver (Vol. I, pp. 835, 836). Certain of them may be mentioned in this connection because of the uniform presence of enlargement of the spleen. Two types of this sort are to be distinguished. In the first, the *acquired*, or *Hayem*[2] type, there is no familial history, the disease may develop at any age, and the course is chronic. There is very decided anemia, chronic acholuric icterus, a large and hard spleen, slight enlargement of the liver, and increased fragility of the red blood-corpuscles. Exacerbations of the condition are frequent, during which there may be very decided temporary decrease in the number of red cells in the blood.

The second, the *familial, congenital*, or *Minkowski*[3] type, is distinguished by the very decided familial nature. It is observed oftenest congenitally or in the new born, but may not appear even for several years. The jaundice is chronic, moderate, and acholuric in nature, the urine containing urobilin in large amount, but no biliary coloring-matter. Frequent exacerbations occur from time to time in which the jaundice grows more intense and the number of red blood-cells diminishes greatly. The fragility of the red blood-corpuscles is increased, the spleen is enlarged, the liver not involved, the anemia is moderate. The general condition of the patient is little if any affected.

Both types of the disorder are to be distinguished from Banti's disease by the fragility of the red blood-corpuscles, and in the Minkowski type by the familial character and usually early development. The prognosis is favorable as to the continuance of life, especially in the familial type; but unfavorable for the recovery from the symptoms. On the ground that the disease is dependent, at least in part, upon increased hemolysis occurring in the spleen, removal of the organ has been recommended, and favorable results have been reported. It is not, however, to be advised unless the general health is suffering.

MORBID GROWTHS OF THE SPLEEN

These are of very exceptional occurrence and of anatomical interest only. Sarcoma is that oftenest seen, being either primary, or more frequently secondary. Fibroma has been reported, and cysts, either dermoid, simple, or echinococcic, may occur.

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[1] Arch. of Pediat., 1914, XXXI, 340.
[2] Presse méd., 1898, I, 121.
[3] Verhandl. d. Kong. f. inn. Med., 1900, XVIII, 316.

CHAPTER III

DISEASES OF THE LYMPHATIC GLANDS AND VESSELS

There exists in early life a very notable predisposition to enlargement of the lymphatic glands. This may be secondary to some disease elsewhere, the glands affected being usually those into which the lymphatics from the diseased regions drain; but not infrequently it is dependent upon a chronically debilitated condition of the general health, and the tendency is often seen also in children who appear in other respects to be entirely well. Certain children, especially some of those formerly classed as scrofulous, but more properly diagnosed in this respect as "lymphatic," exhibit a special proneness to glandular involvement. Certain families, too, have this same predisposition, and this may be inherited. The condition denominated the *lymphatic diathesis* has been described elsewhere (Vol. I, p. 632).

HODGKIN'S DISEASE

(Pseudoleukemia; Adenie; Lymphadenoma; Anæmia lymphatica)

This malady was first described by Hodgkin in 1832;[1] and inasmuch as none of the numerous titles applied is without objection, the name of Hodgkin's Disease may still well be used to designate it.

Etiology and Pathology.—The affection is an uncommon one, and its course and nature not well determined. It is seen with relative frequency in childhood, 17 out of 86 cases collected by Longcope[2] having been under 10 years of age. The youngest patient in a series of 43 cases in children reported by Thursfield[3] was just over 3 years of age. It is decidedly more common in males. The causes and nature are unknown. Some inflammation in the mouth or throat sometimes precedes the glandular swelling, or the disease follows some infectious malady. Tuberculosis was brought into prominence as a probable cause especially by Sternberg,[4] but has latterly failed of corroboration. The theory of the disease as an acute infection has some support, and various bacteria have been described, especially certain Gram-positive bacilli by Much and Fränkel,[5] Bunting and Yates,[6] and others, but none as yet has been proven to be pathogenic.

Pathological Anatomy.—There is a hyperplasia of all the lymphoid tissues of the body, particularly of the lymphatic glands, spleen, liver and lungs, but found also in the lymphatic tissue of the alimentary canal, bones, skin and other regions. The superficial glands everywhere are involved, and the internal ones as well. They may be as large as an egg or larger, and often great masses of these compress the vessels of the neck, the trachea, bronchial tubes or the ureters. They generally remain discrete, and the process does not invade the contiguous tissue. The glands are at first elastic and soft; later firm and hard. On section the substance is pinkish-grey or greyish-white, smooth, with bands of fibrous tissue but without evidence of caseation or suppuration. The

[1] Med.-Chirurg. Transac., 1832, XVII, 68.

[2] Osler and McCrae, Mod. Med., 1915, IV, 755.

[3] Garrod, Batten, and Thursfield, Diseases of Children, 1913, 552.

[4] Zeitschr. f. Heilk., 1898, XIX, 21.

[5] Münch. med. Woch., 1910, LVII, 685.

[6] Arch. Intern. Med., 1913, XII, 236. Journ. Amer. Med. Assoc., 1913, LXI, 1803; 1914, LXII, 516.

spleen is enlarged in 75 per cent. of the cases, but is usually not of such great size as in leukemia, and very frequently shows the presence of lymphoid foci of varying dimensions. Similar deposits are seen in the liver, which is often increased in size; around the bronchi; beneath the pleura; and in the kidneys.

Histologically, the work of Reed[1] shows in the lymphatic glands proliferation of the endothelial cells and of those of the reticulum; lymphoid cells; uninuclear and multinuclear giant cells which differ from those seen in tuberculosis; and later in the case decided increase of connective tissue. Large numbers of eosinophilic cells are present. The structure of the masses in the spleen and elsewhere is of the same character.

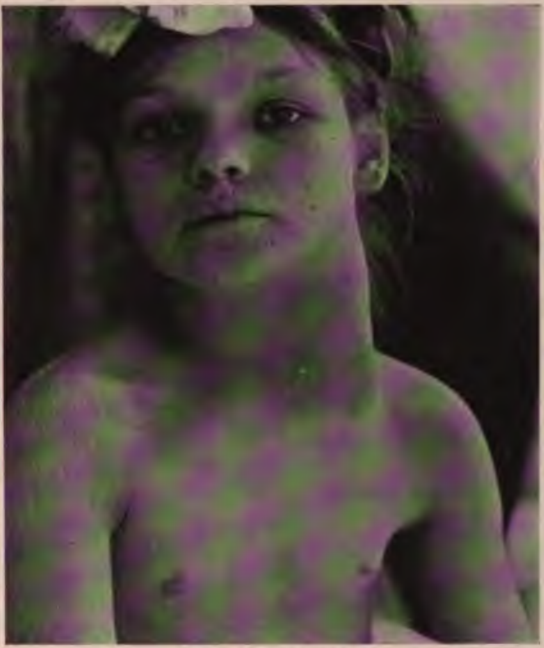

FIG. 402.—HODGKIN'S DISEASE.

Child of 10 years in the Children's Ward of the Hospital of the University of Pennsylvania. Swelling of the glands of the neck, especially of the left side, had lasted 2 years, spleen enlarged, gland excised and microscopical examination confirmed the diagnosis.

Symptoms and Course.—The onset is usually slow, the first symptom being enlargement of some group of glands, much oftenest those on one or both sides of the neck. The swelling is at first soft, later hard; not adherent to the skin or the tissues beneath, and not tender or painful (Fig. 402). The aggregation of numerous glands in close juxtaposition may produce a tumorous mass the size of a fist or larger. The lymphoid tissue of the tongue and throat may also show hyperplasia. Generally months or years pass before any further extension occurs, and then a progressive enlargement of the glands in other regions of the body will be noticed; the axillary glands usually being involved before the inguinal. The spleen and liver exhibit enlargement on palpation. Very rarely lymphomatous growths appear in the skin.

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[1] Johns Hopk. Hosp. Rep., 1902, X, 133.

Early in the case the general condition of health is little if at all affected; but later secondary symptoms appear. One of these is anemia, although the red blood-cells do not often fall below 2,000,000. There is no constant change in the leucocytes. Oftenest there is a polymorphonuclear leucocytosis; sometimes a leucopenia; occasionally an eosinophilia. It has been claimed that the platelets are constantly increased in number (Bunting).[1] Fever of an irregular type is present in most cases; sometimes continuous, sometimes with periods of intermission. Pressure-symptoms of the most varied kind are liable to develop, their nature depending upon the region of the body where overgrowth of lymphatic tissue occurs. Thus there may be edema and pain in the arms from pressure upon the vessels and nerves in the axilla, or the lower limbs may be affected in a corresponding way by the enlarged inguinal glands; cough, dyspnea or dysphasia may be caused by hypertrophy of the mediastinal glands; jaundice by interference with the discharge of bile; or disturbance of urination by pressure on the ureter. Among other symptoms sometimes present are albuminuria, pleural or pericardial effusion, rapid action of the heart, pigmentation of the skin, pruritus, boils, and tenderness along the course of the bones. As the disease advances the masses of glands may reach an enormous size; the spleen may become greatly enlarged; there is a progressive cachexia and emaciation; and death occurs from exhaustion, amyloid degeneration, pneumonia, or as a result of the pressure somewhere in the body.

Course and Prognosis.—An average case can last 2 or 3 years before the fatal issue takes place, although in some instances the course is much longer and in others may be very rapid, continuing only a few weeks or months. Intermissions in the progress of the disease are of common occurrence, during which the glandular swelling diminishes, fever ceases, and all the symptoms improve. These periods may last a month or more, and then relapse occurs. The course of the disease may be delayed by treatment, but the ultimate prognosis is entirely unfavorable. It is doubtful whether any cases have recovered.

Diagnosis.—This rests upon the general lymphatic hyperplasia, without characteristic alternation of the blood. The disease is first of all to be distinguished from *tuberculosis*. There is no doubt that there may occur a general chronic tuberculous adenitis, which is very difficult of diagnosis from Hodgkin's disease; and certainly early in the course of the latter affection the diagnosis from cervical tuberculous adenitis is not easy. In fact, as stated, it has been frequently claimed that Hodgkin's disease was in reality a tuberculous manifestation. The histological structure of the glands in the two diseases is, however, different, and in doubtful cases a gland should be excised and a microscopical study made. Independent of this study is the absence in Hodgkin's disease of the matting together of the glands and of any tendency to suppurate, and the final presence of a much wider diffusion of the process throughout the body. The employment of the cutaneous or other tuberculin reaction is of value if it results negatively. *Lymphocytic leukemia* may produce enlargement of the spleen and lymphatic glands similar to that of Hodgkin's disease; but an examination of the blood will remove all doubt, for even in the unusual cases of leukemia when the total number of leucocytes is not increased, the remarkable relative excess of lymphocytes is characteristic. The occurrence of the hemorrhages seen in leukemia is very infrequent in Hodgkin's disease. *Lymphosarcoma* is readily confounded with Hodgkin's disease. It is to be distinguished by the penetration of the process into the tissues outside of the glands and by the absence of enlargement of the spleen. Histological examination of an excised gland may be required to decide the question. The general enlargement of the lymphatic glands which occurs in some children with the *lymphatic diathesis* or in the course of some other disorders is of very moderate degree, and does not exhibit the progressive extension which is characteristic of Hodgkin's disease. Such a condition could cause no difficulty after serious consideration.

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[1] Johns Hopk. Hosp. Bull., 1911, XXII, 114; 369.

Treatment.—The employment of arsenic and of the x-ray should certainly be tried, since the progress of the condition may be retarded in this way. General tonic and hygienic treatment is also indicated. Removal of the glands early in the case may be tried, on the ground that the disease seems to advance from the original site to others. Treatment, however, is useless in the hope of doing more than prolonging life for a time. Removal of the affected gland is liable to be followed by a return of the disease in greater force. Benefit is claimed from the injection of a vaccine prepared from the microörganisms obtained from the affected glands (Billings and Rosenau;[1] Hatcher and Lemmon).[2]

ACUTE SIMPLE ADENITIS

By this term is designated an acute inflammation of lymphatic glands not dependent upon tuberculosis.

Etiology.—The marked susceptibility which all children have to glandular enlargement renders the frequency of adenitis, acute or chronic, very great, and the variety of causes large. The influence of heredity is very decided, and several children of a family may exhibit a tendency to the process, or the parents may have shown the same; this, too, without there being symptoms which can place the subjects in the domain of the lymphatic diathesis. (See Vol. I, p. 632.) Although common at any period of childhood and much oftener encountered then than later, acute adenitis is seen especially in the first 2 years of life. An exception is the inflammation observed in the course of infectious diseases, which naturally occurs particularly at the school-age. Among these are particularly scarlet fever, measles, rubella, mumps, grippe and diphtheria. A lowered condition of the health is a powerful predisposing cause. Any local peripheral irritation also tends to be followed by inflammation of the glands into which the lymphatics of the region drain. Thus, for instance, an affection of the mouth or throat produces enlargement of the glands below the jaw; one of the scalp or ear is followed by involvement of the occipital, posterior auricular, or superficial cervical glands; that of the finger or arm may result in inflammation of the axillary glands; that of the lower extremities or genital organs affects the glands of the femoral region or groin; and disorders of the intestine produce enlargement of the mesenteric group. The action of bacteria or of toxic substances is the cause in most of these cases, and the glands act as a sieve in restraining the germs from reaching the general circulation. Any of the pyogenic bacteria may be the active agent, but oftenest the staphylococcus or streptococcus.

Pathological Anatomy.—The process is at first a hyperplasia of the lymphoid and connective tissues. The gland is enlarged in size, con-

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[1] Journ. Amer. Med. Assoc., 1913, LXI, 2122.

[2] Journ. Amer. Med. Assoc., 1915, LXV, 1359.

Early in the case the general condition of health is little if at all affected; but later secondary symptoms appear. One of these is anemia, although the red blood-cells do not often fall below 2,000,000. There is no constant change in the leucocytes. Oftenest there is a polymorphonuclear leucocytosis; sometimes a leucopenia; occasionally an eosinophilia. It has been claimed that the platelets are constantly increased in number (Bunting).[1] Fever of an irregular type is present in most cases; sometimes continuous, sometimes with periods of intermission. Pressure-symptoms of the most varied kind are liable to develop, their nature depending upon the region of the body where overgrowth of lymphatic tissue occurs. Thus there may be edema and pain in the arms from pressure upon the vessels and nerves in the axilla, or the lower limbs may be affected in a corresponding way by the enlarged inguinal glands; cough, dyspnea or dysphasia may be caused by hypertrophy of the mediastinal glands; jaundice by interference with the discharge of bile; or disturbance of urination by pressure on the ureter. Among other symptoms sometimes present are albuminuria, pleural or pericardial effusion, rapid action of the heart, pigmentation of the skin, pruritus, boils, and tenderness along the course of the bones. As the disease advances the masses of glands may reach an enormous size; the spleen may become greatly enlarged; there is a progressive cachexia and emaciation; and death occurs from exhaustion, amyloid degeneration, pneumonia, or as a result of the pressure somewhere in the body.

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[1] Johns Hopk. Hosp. Bull., 1911, XXII, 114; 369.

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ACUTE SIMPLE ADENITIS

By this term is designated an acute inflammation of lymphatic glands not dependent upon tuberculosis.

Etiology.—The marked susceptibility which all children have to glandular enlargement renders the frequency of adenitis, acute or chronic, very great, and the variety of causes large. The influence of heredity is very decided, and several children of a family may exhibit a tendency to the process, or the parents may have shown the same; this, too, without there being symptoms which can place the subjects in the domain of the lymphatic diathesis. (See Vol. I, p. 632.) Although common at any period of childhood and much oftener encountered then than later, acute adenitis is seen especially in the first 2 years of life. An exception is the inflammation observed in the course of infectious diseases, which naturally occurs particularly at the school-age. Among these are particularly scarlet fever, measles, rubella, mumps, grippe and diphtheria. A lowered condition of the health is a powerful predisposing cause. Any local peripheral irritation also tends to be followed by inflammation of the glands into which the lymphatics of the region drain. Thus, for instance, an affection of the mouth or throat produces enlargement of the glands below the jaw; one of the scalp or ear is followed by involvement of the occipital, posterior auricular, or superficial cervical glands; that of the finger or arm may result in inflammation of the axillary glands; that of the lower extremities or genital organs affects the glands of the femoral region or groin; and disorders of the intestine produce enlargement of the mesenteric group. The action of bacteria or of toxic substances is the cause in most of these cases, and the glands act as a sieve in restraining the germs from reaching the general circulation. Any of the pyogenic bacteria may be the active agent, but oftenest the staphylococcus or streptococcus.

Pathological Anatomy.—The process is at first a hyperplasia of the lymphoid and connective tissues. The gland is enlarged in size, congested, edematous, and hard. Microscopical examination shows a lymphoid hyperplasia attended by an infiltration of polymorphonuclear cells. If the infection has been too great for resolution to take place, suppuration occurs. In this event the gland becomes adherent to the surrounding tissue through a secondary inflammation of this. As a rule in suppurative cases only one gland is at first affected, although the inflammation may spread to others of the group. In the adenitis depending upon certain of the infectious diseases, one or more groups of glands may be involved.

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[1] Journ. Amer. Med. Assoc., 1913, LXI, 2122.

[2] Journ. Amer. Med. Assoc., 1915, LXV, 1359.

Symptoms.—These vary with the cause. The cervical glands are the ones attacked far more often than any others, so far as the external lymph-nodes are concerned. The existence of acute inflammation of the internal glands cannot be determined in a manner to permit of conclusions as to its frequency. The first symptom of adenitis, apart from the manifestations of the primary disease, is the development of a hard swelling, movable, little if at all tender, and unaccompanied by fever. This inflammation may persist unchanged during the course of the primary disease, and disappear with it or somewhat later. This description applies to the mildest cases. In those of rather more decided severity, the enlargement of the glands is more rapid and greater, and its duration is independent of the primary process. There is tenderness and slight fever, yet but little other constitutional disturbance. The surrounding tissue is not involved, and there is generally no redness. Slowly the swelling disappears, lasting a number of weeks before the threatening symptoms are over, and often leaving an induration for a considerably longer time. In the suppurative cases the enlargement is progressive; although very often with a stationary period, after which fever develops or increases, reaching 103° or 104°F. (39.4° or 40°C.); the surrounding cutaneous tissue becomes involved in the process; a large, tense, hard, very tender and painful mass is rapidly produced, with the skin over it reddened and shining; and finally, unless relieved by operation earlier, a portion of the surface becomes softer and fluctuating, ruptures, and pus is discharged (Fig. 403). Some cases run a more acute course from the beginning, with severer symptoms; and this is especially true of adenitis in infancy, at which period suppuration is especially liable to occur. In most of the infectious fevers, with the exception of scarlatina, there is little tendency to suppuration. In this disease the glandular enlargement is widespread, although suppuration is liable to occur only in the glands of the neck and below the jaw.

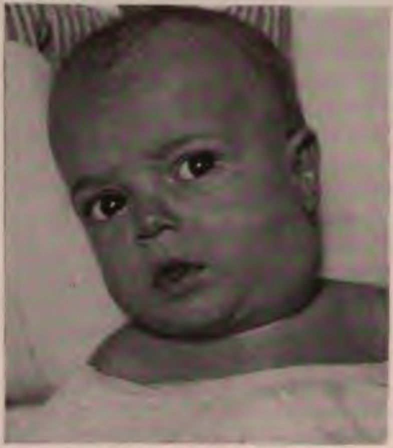

FIG. 403.—SIMPLE CERVICAL ADENITIS. Infant of 5 months in the Children's Hospital of Philadelphia, with suppurative adenitis. Incision later.

Prognosis.—This is favorable for final recovery, but very uncertain as regards the development of suppuration, the only exception being the adenitis seen in the infectious diseases, which seldom goes on to suppuration except in scarlet fever. Of the cases due to other causes the majority occurring in infancy suppurate; but at other periods it is impossible to predict the outcome in this respect. Very often a swollen gland will remain in a quiescent state for a week or more and then take on a rapid increase of severity and suppuration follow.

The duration of adenitis is variable. In the cases which do not suppurate, from a few weeks to a couple of months may be passed before recovery is complete. The suppurative cases generally show signs of this in the 2d or 3d week or earlier. The recovery after the abscess is opened is generally rapid, unless it has been allowed to exist too long before incision had been made.

Diagnosis.—This is a matter seldom presenting difficulty. One of the commonest errors is that of mistaking cervical adenitis for *mumps*. If the adenitis is well-advanced when first seen, and the surrounding tissue implicated, it may for a time be difficult to come to a positive conclusion. However, the fact that the center of swelling in mumps is over the parotid gland, and in adenitis is below or at the angle of the jaw, will aid in distinguishing between the two. A still greater problem is that of differentiating between simple and tuberculous adenitis. This will be discussed when considering the latter affection.

Treatment.—Preventive treatment consists in maintaining and improving the general health and in quickly removing all sources of peripheral irritation, especially those connected with the pharynx, ears, nose and mouth. After adenitis has actually appeared, treatment of the same nature should continue, and efforts should be made to abort the attack if this is still possible. Either cold or very hot applications may aid in this. The employment of a poultice may, it is true, increase the liability to suppuration; but frequently, on the other hand, aids resolution by relieving the congestion of the gland. Ointments of various sorts, particularly of ichthyol or of iodine in some form, seem useful in some cases; and in others gentle massage and rubbing with stimulating liniments is to be recommended. If suppuration is evidently occurring, cold or hot applications will relieve the pain; and just as soon as an area of softening appears, this should be incised and dressed antiseptically. Further surgical procedure is seldom necessary.

ACUTE EPIDEMIC INFECTIOUS ADENITIS

(Glandular Fever)

There is a condition of acute and often epidemic infectious lymphadenitis which was described under the title of "glandular fever" by E. Pfeiffer.[1] Much discussion has taken place since then regarding its existence as an independent affection. I have never been convinced of this, and the disorder has seemed to me to be a symptom merely, appearing usually epidemically, it is true, but in the course of a number of other diseases. Among these is certainly to be included grippe, as well as any epidemic occurrence of inflammation of the nasopharynx of other nature, and probably other causes as well; the characteristic in these cases being that the inflammation of the glands becomes the most prominent symptom. That some general infection has taken place is shown by the occasional development of enlargement of the liver and spleen, as well as of a widespread involvement of the lymphatic glands in various parts of the body. The disorder is best considered a variety of acute, simple lymphadenitis. It is encountered oftenest in infancy and early childhood.

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[1] Jahrb. f. Kinderh., 1889, XXIX, 257.

Symptoms.—The onset is sudden, with high fever, pain on moving the neck, sometimes nausea and vomiting, abdominal pain, and pain in the bones. Some degree of inflammation of the throat and nose is present, but is of secondary importance. Distinct enlargement of the cervical glands is observed after from a few hours up to from 1 to 2 days of illness. This affects especially the glands along the course of the carotid upon both sides, commonly on one side before the other; then later others in the neck, and often with involvement of the axillary and inguinal group as well; sometimes the mesenteric glands, and occasionally with enlargement of the liver and spleen. The glands are tender and swollen, but the skin is not reddened, and suppuration is rare. They increase in size for a day or two, reaching the dimensions of a pigeon's egg or occasionally larger, often smaller. Substernal pain sometimes occurs, supposed to indicate enlargement of the bronchial nodes. The inflammation lasts from 2 to 3 weeks, although the constitutional symptoms disappear earlier, the fever ceasing in from 2 to 3 days. Occasionally a nephritis occurs as a complication. The condition can scarcely be confounded with any other, unless it be an acute lymphocytic leukemia, and the examination of the blood will differentiate here. The shortness of the attack and the complete recovery are characteristic. Treatment other than symptomatic is unnecessary.

CHRONIC SIMPLE ADENITIS

Etiology.—This disorder is common in children. It may remain after one or repeated attacks of acute adenitis in which complete resolution has not taken place; or may be of a chronic nature from the beginning, attending some persistent source of irritation, such as the presence of continued irritation of the scalp by pediculi, chronic otitis media, and the like; and especially the presence of adenoids or of hypertrophy of the faucial tonsils. Any impaired condition of health is a very frequent cause. Consequently chronic enlargement of the glands is especially common in institutions for children, or under any circumstances in which children are crowded, or receive poor food or insufficient fresh air. A part, too, of the symptoms of the lymphatic diathesis (Vol. I, p. 633) is the chronic glandular enlargement which may be present. Syphilis is a very exceptional cause seen usually in later childhood. The active cause is a chronic toxic or mechanical influence, or bacterial involvement of the glands. Any bacteria present are of low virulence, not sufficient to produce suppuration.

Symptoms.—Practically the only symptom is the glandular enlargement. A single gland or oftener a whole group may be involved, or sometimes glands in various parts of the body, the latter condition being seen particularly in instances of the lymphatic diathesis. The glands oftenest affected are those of the neck. They are generally only moderately swollen, hard, movable and not tender. There may be a gradual decrease in size, or no change may be noticed after the first discovery of the enlargement; or there may be observed an alteration in the degree of swelling from time to time. There is no tendency to suppuration, and no constitutional symptoms are produced. The disorder may last for several months and then disappear slowly, or may be very persistent.

Diagnosis.—Although the disease may form a part of the complex of symptoms of the lymphatic diathesis, in other cases it is to be distinguished by the entire absence of other manifestations of this condition.

Lymphatic leukemia produces a more widespread and greater hypertrophy, and is recognized by the examination of the blood. Hodgkin's disease results in a very much greater enlargement and is of an even more chronic nature. It is only very early in the disease that a real difficulty in diagnosis can occur between the two. The chief question to be solved is the distinguishing of simple chronic enlargement from tuberculous adenitis. This will be discussed in the next section.

Treatment.—The first object is the removal of the source of irritation; and, since the disease is oftenest cervical, particular attention should be given to the condition of the mouth, nose and pharynx. In addition measures must be taken to improve the general health. The employment of tonic remedies, especially cod-liver oil, is of service for this purpose, and sometimes of iodide of iron or arsenic; and a change of climate is often most beneficial. Massage of the glands may be useful, or the application of ichthyol or iodine preparations.

TUBERCULOUS ADENITIS

(Scrofula)

(See also under Tuberculosis, Vol. I, p. 557, and the Diatheses, p. 630)

Etiology.—In this very common disease the active agent is, of course, the tubercle bacillus; but predisposing causes are to be considered as well. Children are oftener attacked than adults. In infancy the internal glands, such as the tracheobronchial and the mesenteric, are those usually affected, while in childhood and later the disease most frequently involves the external glands, especially the cervical groups. In 300 cases of the disease in this situation examined by Fraser[1] 10 per cent. occurred in the 1st year and 40 per cent. from 2 to 5 years. Children born of tuberculous parents have an undoubted inherited tendency to develop tuberculous adenitis, and the same is true of subjects of the exudative diathesis and of lymphatism. (See Vol. I, pp. 630 and 632.) Tuberculous adenitis is also liable to occur as a sequel to attacks of the infectious disorders, especially pertussis and measles; and diseases of the bronchial mucous membranes, or of the intestines, are readily followed by tuberculosis of the tracheobronchial or the mesenteric glands respectively. A gland attacked by a simple inflammation may become tuberculous later.

Pathological Anatomy.—In the more rapid cases the gland is swollen, hyperemic, and on section of a pink color. Soon, however, it grows harder, and the section reveals scattered minute greyish spots, the foci of tuberculous infiltration; and still later by fusion of these foci the whole section becomes greyish, and finally the gland caseates. Meantime the surrounding tissue grows inflamed and adherent, and a number of glands of the group are matted together. Aided by the softening process an abscess may be produced; or the gland may remain caseous and be encapsulated by fibrous tissue; or it may be replaced chiefly by connective tissue. Histologically the change consists of the production of tubercles (see Vol. I, p. 544), with hyperplasia of the cellular elements of the gland, together with infiltration by leucocytes. Tubercle bacilli are, as a rule, not numerous, and are found chiefly in the early stages, and in a thoroughly caseous gland it may be difficult to discover any. The pus of a suppurating gland is sterile in uncomplicated cases, and is not, strictly speaking, *pus*, but rather a cheesy material from the broken down tubercles and glandular tissue. The process is, however, often complicated by the presence and action of pyogenic organisms, and a genuinely purulent substance results.

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[1] Clin. Journ., 1915, XLIV, 41.

This description applies to the cases of adenitis with the more rapid course. In a second group the duration is more prolonged, and the glands are less tender and hyperemic; hard and with thickened capsules; and on section show histologically a great increase of the connective-tissue elements, this acting as a conservative process. Foci of caseation may be seen surrounded by the fibrous overgrowth. These glands are not so liable to be matted together or to be bound to the adjacent tissues, and suppuration is not likely to take place.

The most favorable final process in tuberculous adenitis is the production of cicatricial connective tissue and final absorption of the tuberculous products. Another favorable outcome is the encapsulation of caseous masses by the fibrous tissue; these remaining caseous or finally less often becoming calcified. Without this encapsulation, the caseous masses will probably advance to the more unfavorable termination; viz. suppuration. They then rupture externally; or into a lymph-vessel or blood-vessel and produce tuberculous meningitis, tuberculous bronchopneumonia, or general miliary infection.

Regarding the relative frequency of tuberculosis in the different glands of the body: of the external glands those of the neck are the ones affected in the great majority of instances, and usually these alone. In 155 examples of tuberculosis of the external glands reported by Treves[1], those of the neck were involved in 145, and these alone in 131; those of the axilla in 17, and those of the groin in 8. Any of the groups in the neck may be attacked, although the site of predilection is the deep cervical or the submaxillary glands. The frequency of tuberculosis of the tracheobronchial and mesenteric glands can be determined only in cases coming to autopsy. Enlargement of the former is nearly always present in fatal cases of tuberculosis in early life. (See p. 545.)

Classification and Symptoms.—For convenience of study of clinical symptoms, tuberculous adenitis may be divided into (1) Generalized tuberculous adenitis; (2) Localized tuberculosis of the external glands; (3) Tuberculosis of the tracheobronchial glands; and (4) Tuberculosis of the mesenteric glands.

1. Generalized Tuberculous Adenitis.—This is an uncommon affection in which tuberculosis develops in nearly all the lymphatic glands of the body. It may appear without warning in children previously in good health, or oftener may be seen in those already debilitated by unhygienic conditions or by some preceding acute disease; or it may come on suddenly as a sequel to some localized tuberculous process. The onset of the attack is sudden, with malaise, loss of appetite, fever and emaciation. The glands rapidly hypertrophy, either simultaneously or oftener successively, and may reach a large size filling the sides of the neck, the axillæ and the groins. At autopsy widespread involvement of the internal lymphatic glands also is discovered, and sometimes the enlargement is found to be chiefly here.

2. Localized Tuberculosis of the External Glands.—This disease is by much the most frequent in the cervical glands, those of them attacked depending upon the lymphatics through which the bacteria enter. The infection is generally derived from the nasopharynx. Less frequently it is an ascending infection from tuberculous tracheobronchial glands. Involvement of the cervical glands is one of the forms of tuberculosis oftenest dependent upon the bovine type of bacillus. (See Vol. I, p. 542.) Most frequently affected are the deep cervical and the submaxillary groups and those below the parotid glands. The first **symptom** noted is the presence of small, hard nodules, often called "kernels," in the neck, appearing at the angle of the jaw, or along the anterior or posterior border of the sternocleidomastoid muscle. The swelling has come on gradually and without any constitutional disturbance. Usually the enlargement is present on both sides, although more on one than the other (Fig. 404). The glands are not tender and appear to be distinct from one another. Further increase in size goes on slowly, and the fact that there are a number of glands involved becomes very evident. They are finally no longer distinctly separated from each other, and some tenderness may develop. The skin is freely movable over them. The degree of enlargement varies from time to time, any condition which lessens the general health being liable to increase the swelling. If the process advances to softening and discharge, the skin over the swelling becomes adherent; the surrounding tissue is swollen and indurated; the individual glands can no longer be outlined; and an abscess forms with softening, and finally discharges itself on the surface of the neck and leaves a sinus, which heals only after all the glandular tissue has broken down and been evacuated and results in much disfigurement (Fig. 405). In this way one gland after another may break down and discharge, either through the old opening or oftener through a new one, and the process is often long drawn out. During the continuance of active in-

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[1] Scrofula and Its Gland Diseases, 1882, 123.

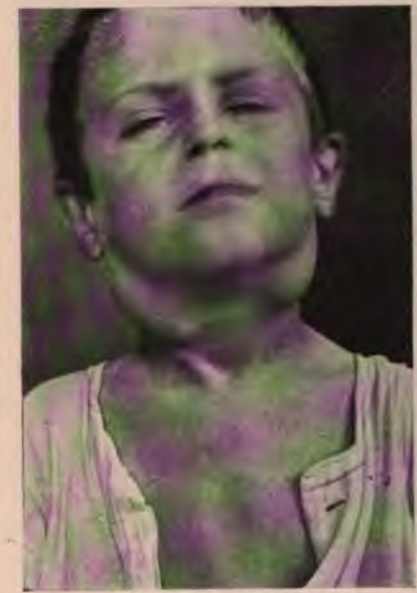

FIG. 404.—TUBERCULOUS ADENITIS. (*Courtesy of Dr. H. R. Wharton.*)

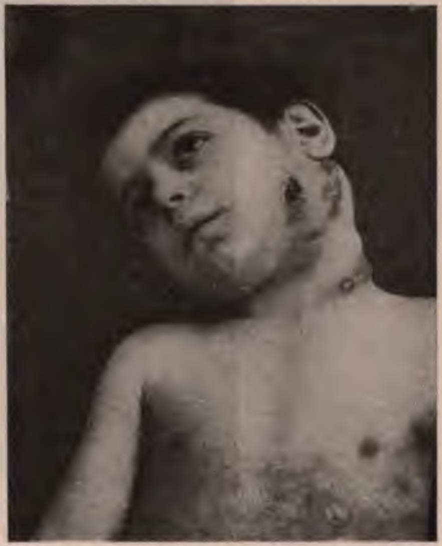

FIG. 405.—TUBERCULOUS CERVICAL LYMPH-NODES.

A neglected case. (*Dowd, Annals of Surgery,* 1905, *XLII.*)

flammation, when abscess is forming, the patient has fever and the general health suffers.

Not all tuberculous glands undergo this course, and resolution can take place; or the glands remain indurated and adherent to each other without suppurating. Tuberculous infiltration may extend from the cervical glands to those just above the clavicle, the posterior part of the neck, or the axilla; or this last-mentioned region or the groin may exceptionally be the first or the only part affected.

Apart from the presence of the enlarged glands, and not caused by this, many children show a group of symptoms to which the title *scrofula* has often been applied. These children are anemic, flabby, and stupid in appearance. There is a tendency to blepharitis causing reddened, crusted, thickened eyelids; ulcerative or phlyctenular keratitis is common, leading to photophobia and corneal opacity; constant thick discharge from the nose is frequent, producing swelling and redness and often eczema of the upper lip; and chronic purulent otitis media is common (Fig. 406). The teeth are often carious, and obstinate cutaneous lesions may occur. Whether all the lesions referred to depend upon the actual presence in them of tubercle bacilli, or whether the mere existence of such bacilli elsewhere in the body, oftenest in the tracheobronchial glands, brings about in some way, presumably through a toxin, an unhealthy state of the tissues in general; or whether again some of these symptoms belong rather to the exudative-lymphatic diathesis in combination with a complicating tuberculosis, has been a matter much discussed and concerning which diverse views are entertained. It is certain that tuberculous adenitis is repeatedly observed without the attendance of any such "scrofulous" symptoms, and it would seem very probable that

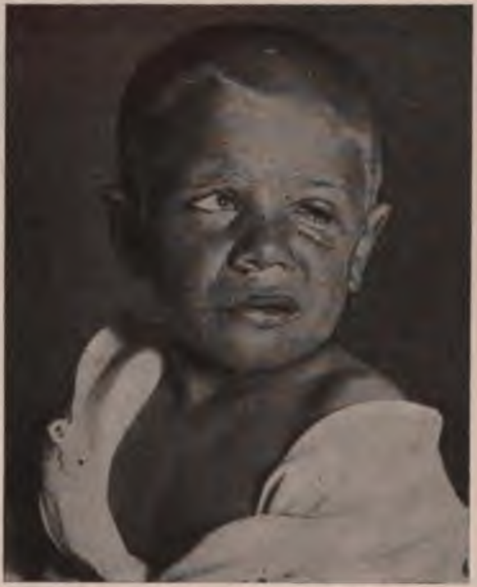

FIG. 406.—THE "SCROFULOUS" FACE.

Showing the inflamed eyes, thickened alæ and irritated upper lip. (*von Pirquet, in Feer's Lehrbuch der Kinderheilkunde*, 1914, 671.)

the lesions, which are tuberculous, occur in certain individuals because the existence of the diathesis referred to produces a lack of resisting power in cases where infection by the tubercle bacilli has occurred. Inasmuch, then, as the lesions of "scrofula" are those of tuberculosis, it seems better to do away with the term entirely and to regard the complex of symptoms as one of tuberculosis occurring in a subject with an exudative-lymphatic diathesis.

3. Tuberculosis of the Tracheobronchial Glands.—(See also Tuberculosis, Vol. I, p. 556.) The glands which may be affected are not only the groups about the trachea or the large bronchial tubes, but the chains extending along the smaller bronchi into the lungs (Vol. I, Fig. 181, p. 555). Generally all the groups are involved, but not all to the same extent. The great frequency of tuberculosis of these glands in early life, as shown at autopsy, has already been alluded to (Vol. I, p. 545). It is undoubtedly one of the most common forms of tuberculosis found at autopsy upon infants and children. It may exist apparently alone, although probably secondary to an undiscovered pulmonary lesion, but is usually associated with definite clinical pulmonary tuberculosis of some sort; or it may be followed finally by tuberculous meningitis or miliary tuberculosis. A few or, often, many of the glands of the chain show enlargement. This is generally of moderate degree, but sometimes a large mass is produced by the matting together of a number of glands. The disease is most frequently situated chiefly upon the right side. Suppuration does not often occur except in infancy, and encapsulation with caseation or calcification is the usual outcome, the glands lying dormant, although always a source of danger as the focus for a more widespread infection of the lungs or other parts of the body.

Clinically, however, tuberculosis of the bronchial lymph-nodes is not a common disease. General *symptoms* are absent altogether or are very indefinite. They consist in the gradual development of deterioration of health, shown by loss of appetite and of flesh, pallor, and low irregular fever; all without discoverable cause. Sometimes the onset is more sudden and the fever higher. This is particularly true if suppuration is occurring. None of these symptoms can be called characteristic, since they could be produced by tuberculosis elsewhere in the body. Local evidences are present only if the glands attain large size and cause local interference, or if an abscess has formed; but such symptoms develop usually only in early and later childhood, infants with the disease dying of other tuberculous manifestations before the glandular ones appear. Indeed, at any time in early life pressure-symptoms are oftener absent than present. In 68 cases examined by Gölz[1] positive evidence of pressure could be discovered in no instance.

A caseous suppurating gland may rupture into a large bronchus or into the trachea and produce asphyxia; or an abscess may break into the mediastinum and penetrate in different directions or open externally, or may perforate one of the large blood-vessels. Cases are reported where the trachea, bronchi, esophagus, vena cava, or pneumogastric or recurrent laryngeal nerve have been compressed by enlarged glands. Interference with the nerve produces one of the most frequent clinical manifestations; consisting in an irritating, hacking, paroxysmal cough suggesting pertussis. It may be absent for considerable periods and then return, depending upon a changing degree of glandular enlargement. It accounts, too, for the production of dyspnea. Pressure on the trachea

[1] Med. Klinik, 1914, X, 194.

may likewise produce a very characteristic noisy cough and prolonged dyspneic, noisy respiration. This comes on in paroxysms, with cyanosis, strongly suggesting asthma or spasmodic croup. Between the paroxysms the stridor is diminished in intensity or even disappears. Pressure upon the large veins may give rise to a murmur audible over the upper part of the sternum when the head is thrown back. This, the "Eustace Smith sign" is of some value when present, but is often absent; and may occur in other conditions. If the compression is great, there may be cyanosis and edema of the face and dilatation of the superficial veins. Among other physical signs to be sought for, if the mass is of large size, is dullness on percussion posteriorly beside the spinal column over the roots of the lungs between the 3d and the 6th dorsal vertebræ. This sign is, however, often absent. In front there may be dullness over the upper part of the sternum, but it is difficult to distinguish this from that caused by an enlarged thymus. If one main bronchus is compressed, the lung on this side may give a percussion-note of diminished resonance. Percussion dullness over the apices of the lungs, or in the spinous fossæ, supposed to be dependent upon pulmonary involvement, may be the result of the pressure of enlarged tuberculous bronchial glands. Auscultation is sometimes of value. Posteriorly there may be tracheal or bronchial respiration along the spine; while a still earlier change is a spoken or whispered pectoriloquy between the 7th cervical and the 1st dorsal vertebræ, and sometimes as far downward as the 4th or 5th dorsal, generally most marked on the right side (D'Espine's[1] sign). At this point the tracheal voice-sound should normally change into that of the lung, and a continuation of this character below the point mentioned is abnormal. The value of the sign is, however, very uncertain, on account of the discrepancies in the statements of investigators as to the exact position at which the change should take place. Morse,[2] after the examination of over 600 children, places it at the 7th cervical and 1st dorsal. Howell,[3] examining approximately an equal number, found it oftenest at the 2d or 3d dorsal. It would be at least safe to assume that the presence of pectoriloquy at or below the 3d dorsal vertebræ is an indication of enlargement of the bronchial glands. It does not prove, however, that this is of a tuberculous nature. If a main bronchus is compressed, there may be feeble respiration in the clavicular region. Radiography is of service in outlining the presence of a mass within the thorax, if of large size and especially if calcareous. With regard to the diagnosis of tracheobronchial tuberculosis, it must be admitted that in spite of the numerous physical signs which may occur, in the majority of cases no very certain conclusions can be reached, except by the employment of the *x*-ray, and even this must be carefully correlated with other signs and the symptoms.

4. Tuberculosis of the Mesenteric Glands.—Long known as "tabes mesenterica" this condition does not now possess the clinical importance which was attached to it formerly. The frequency varies according to different statistics, but involvement of the glands to a moderate degree, discoverable only at autopsy, would appear to be very common. In 2288 autopsies on tuberculous children reported by different observers and collected and studied by Freeman,[4] involvement of the mesenteric glands was present in from 16 per cent. to 40 per cent. It may occur apparently as a primary affection and perhaps alone, or be clearly secondary to tuberculosis of the intestine; but in the latter event there is no relationship between the degree of the process in the two regions. A very great enlargement of the lymph-nodes, may, perhaps, attend a slight involvement of the intestines, or the converse may be the case. Later investigations have placed the mesenteric adenitis as always secondary to a tuberculous lesion of the intestine at the portal of entry. (See Vol. I, p. 543.) The enlarged glands may be very numerous, discrete, and about ½ inch (1.27 cm.) in diameter; or they may be much larger and fused, forming masses of great size. Resolution or, oftener, caseation takes place; suppuration or calcification is not frequent. Some degree of localized peritonitis is very liable to be present in well-marked cases.

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[1] Bull. acad. de méd., 1907, 3 s, LVII, 167; Brit. Med. Journ., 1910, II, 1136.

[2] Amer. Journ. Dis. Child., 1916, XI, 276.

[3] Amer. Journ. Dis. Child., 1915, X, 90.

[4] Med. News, 1905, May 27.

From a clinical point of view the disease is uncommon. The general *symptoms* are often masked by those indicating or accompanying intestinal ulceration. When this is not the case there is impairment of the general health, with wasting, anemia, loss of appetite and strength, irregular fever, and slight abdominal pain. The emaciation may finally become extreme. Tympanitic distention of the abdomen and the occurrence of diarrhea or constipation belong rather to symptoms of intestinal tuberculosis than to those of the mesenteric involvement; or the distention may indicate tuberculous peritonitis.

The most characteristic of the local symptoms is the discovery by palpation of the enlargement of the glands. This, as a rule, cannot be made, on account either of the moderate degree of hypertrophy or of the abdominal distention. In other cases enlarged glands can be distinctly felt in the deeper parts of the abdomen near the spinal column, or, in cases of unusually great hypertrophy, close to the abdominal walls. Rectal examination may aid in revealing the tumors. Very exceptionally other local symptoms of different sorts may result from pressure of the growths upon internal organs, such as the portal vein, the vena cava or the thoracic duct; or there may be intestinal obstruction, or ulceration into arteries. An error in diagnosis which has repeatedly occurred is the mistaking for an appendicitis an acute tuberculous inflammation of the glands near the cecum (Gage).[1]

Course and Prognosis of Tuberculous Adenitis.—This naturally varies with the seat of the inflammation. In *generalized tuberculous adenitis* the course is unfavorable, death taking place from increasing cachexia or some complicating tuberculous condition of other parts. This occurs often after an illness of only a few weeks or months, although sometimes the duration is longer and a somewhat more chronic form of the disease develops. In exceptional cases recovery takes place.

The course of *cervical adenitis* is usually much prolonged and the duration uncertain, one gland after another breaking down and being discharged, often with the elapse of months between these occurrences. There is also the possibility of different groups of glands becoming involved, the infection spreading from the carotid group to those about the clavicle and even downward to the intrathoracic group. The more favorable cases terminate in resolution after several months. In how many instances suppuration will take place is also uncertain, as it is how soon this result will follow, and the statistic of writers are at variance. Treves[2] found it occurring in 93 out of 131 instances of cervical adenitis.

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[1] Boston Med. and Surg. Journ., 1915, CLXXIII, 301

[2] *Loc. cit.*, 150.

The duration of the inflammation before suppuration develops averaged 3½ years in Treves series,[1] the minimum being a few months and the maximum 12 years. In many the affected glands are quiescent for several years and then produce pus more or less rapidly. Neglected suppurating cases will retain a discharging sinus for long periods, or leave ulcers which are most resistent to treatment, and finally heal with irregular and very disfiguring scars. The prognosis for life is favorable; yet apparently not to the extent commonly believed. Most statistics show but few cases in which the process has extended to other regions of the body beyond the glands. The prognosis of tuberculosis of the axillary and supraclavicular glands is not quite so favorable, as there is greater danger of the involvement of the pleura or lung.

As regards the local prognosis of suppurating glands, the earlier operation is done and the more thorough the removal of dead glandular tissue, the shorter usually will the duration of the process be, the less the liability of recurrence, and the less scarring will result.

In *tuberculosis of the tracheobronchial glands*, the duration cannot be estimated, because the time of onset of the enlargement cannot be determined. Even after symptoms have justified a provisional diagnosis, the course is subacute or chronic. It is certain that the inflammation may be very long-continued, and that recovery may take place. There is always, however, imminent danger of the extension of the tuberculous process to other regions by way of the blood-vessels, and this is particularly true under the age of 6 years, at which time softening of the glands is liable to occur. Always, too, there is the danger of rupture of an abscess into the pericardium or pleura, or the escape of pus into the trachea, with a sudden, fatal asphyxia resulting.

The course of *tuberculosis of the mesenteric glands* is very similar to that of the disease situated in the bronchial group. Clearly it may continue for years without symptoms; and even with symptoms present the condition may last for months, with death finally from exhaustion. Yet occasionally disappearance of the symptoms may take place even with palpable tumors remaining.

Diagnosis.—The diagnosis of the first two classes of tuberculous adenitis described generally presents little difficulty. That of the other two is far from easy and often only provisional. *General glandular tuberculosis* is to be distinguished from leukemia, lymphosarcoma and Hodgkin's disease. The first is readily recognized by an examination of the blood; the second by the occurrence in one group of glands only, at least at the onset; the third by the absence of evidence of inflammation and of the fusion of the glands which is seen in tuberculosis, the slower onset and longer course, and the histological and bacteriological character of a gland excised for study. The diagnosis of *cervical tuberculous adenitis* is to be made from simple acute or chronic adenitis and from Hodgkin's disease. From the acute adenitis the diagnosis is generally not difficult. Tuberculous adenitis in general runs a slower course; the glands on both sides of the neck are enlarged; a whole group is more or less involved; there is more matting together of the glands; softening is very much slower in taking place, suppuration not occurring for at least a number of months, and the pus which forms is of a more cheesy character. Cases of simple adenitis which do not resolve go on to suppuration in a few weeks. The absence of a tuberculin reaction speaks strongly for simple adenitis. Simple chronic adenitis gives more diagnostic trouble, yet here, too, there is no fusion of the glands or adherence to the overlying skin; softening and suppuration do not occur; the duration is shorter; a discoverable irritating cause is often present, and the tuberculin test is negative unless there is concealed tuberculosis elsewhere in the body. It must be admitted, however, that not infrequently the distinction between the simple chronic and the tuberculous forms may at times be difficult or impossible until after excision has been done and a microscopical study made. In Hodgkin's disease the course is very slow; caseation or suppuration does not occur; groups of glands elsewhere than in the neck are involved at the same time or in succession; and the constitutional symptoms are eventually more severe.

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[1] *Loc. cit.*, 151.

The diagnosis of *tracheobronchial tuberculous adenitis* is one of great difficulty unless local symptoms develop, and can be made only by exclusion. The combination of irregular, never continuous fever, generally of mild degree, and extending over a long time, with wasting, anemia, malaise, fretfulness, loss of appetite and similar symptoms of failing health is strongly suggestive in the absence of any other discoverable cause. The most characteristic of the local symptoms are the paroxysmal cough and the attacks of dyspnea suggesting croup or asthma. The cough is like that of pertussis, but is seldom attended by vomiting and does not possess the whoop. The dyspnea is generally more or less constant, but with paroxysmal exacerbations. The physical signs obtained by percussion, auscultation and radiography have already been described. They can be regarded only as corroborative, since the pressure-symptoms detailed are often very misleading and by no means unequivocal. A negative result of the tuberculin reaction is strongly against the existence of tracheobronchial tuberculous adenitis. The diagnosis of *tuberculosis of the mesenteric glands* is even more uncertain. In the absence of local manifestations, the principal diagnostic signs are the progressive wasting and debility, frequently of extreme degree and without discoverable cause, combined with irregular fever and often with a complicating disturbance of the intestinal functions. Of the local manifestations the most characteristic is the glandular enlargement in the abdominal cavity; and if this is present along each side of the spine the diagnosis is fairly certain. Yet, the possibility of the occurrence of fecal masses, hydatids, tuberculous peritonitis, new growths, or subacute intussusception is to be considered. In tuberculous peritonitis the masses are generally numerous and nearer the abdominal walls; but involvement of the peritoneum is very likely to be a complication of the disease of the mesenteric glands. Fecal masses can be excluded by the results of treatment; new growths, such as renal sarcoma, can be detected by their position and their history. Yet it is well in all cases of what appear to be new growths in the abdominal cavity to entertain seriously the possibility of their being in reality tuberculous mesenteric glands.

Treatment.—This depends to some extent upon the situation. In all forms of tuberculous adenitis especial care should be given to the upbuilding of the general health by tonic remedies, including cod-liver oil and iodide of iron; abundant digestible food; out-door life, and change of climate. This has already been discussed under Tuberculosis (Vol. I, p. 561). The avoidance of all infectious and other acute diseases is important, since under their influence the tuberculous process is very likely to grow worse. Special symptoms demand symptomatic treatment, such as the control of fever when unusually high, and the allaying of cough when there is involvement of the bronchial glands. In the case of tuberculosis of the cervical glands, it is important to remove all such sources of local irritation as carious teeth, chronic otorrhea, ulcers in the mouth, and the like; or those situated in the tonsillar tissue of the pharynx or nasopharynx. Retrogression of the inflammation of the glands may follow this. The question of operative treatment is to be considered. An opportunity for resolution should certainly be given, with careful watching maintained that the condition does not grow worse. Then, after a delay of not more than a few months, if improvement has not commenced, a thorough removal of all the enlarged glands discoverable is to be advised. It is important to operate, if possible, before the glands become adherent to the surrounding tissues, otherwise the recovery will be much delayed, and the scarring will be greater. The scar which results after an operation properly timed will finally become very little noticeable, provided that in making the incision all due attention has been paid to the natural folds in the skin of the neck. It is to be noted that some surgeons do not favor this complete removal and prefer other methods, among them small incisions with the use of Bier's hyperemia. The value of tuberculin has been much disputed. From a survey of the literature and personal experience in 191 cases of tuberculous adenitis in children, Waugh[1] concluded that there is no satisfactory proof of its value, and that there can be none because a positive diagnosis can be made only by examination of a gland after excision. The value of the Roentgen ray in promoting absorption of tuberculous glands is still under discussion. There is no question but that the treatment is able to accomplish good results in many cases, and many physicians are strongly in favor of it.

If suppuration has already occurred the gland may be either opened by a small incision, or the thorough extirpation of the whole group practised, provided so many adhesions have not already formed that this is impossible.

DISEASES OF THE LYMPHATIC VESSELS

A diffuse *lymphangiectasis* is rarely seen. It may be either congenital or acquired. In tropical climates lymphangiectasis may result in an *elephantiasis* depending upon the presence of filaria. In other regions elephantiasis may be produced by inflammation of the lymphatic vessels. It is occasionally seen in children. Affections of the *thoracic* duct are very rare, the most frequent being the production of chylous ascites or chylous pleural effusion, depending upon some damage of the duct by tumors or in other ways. (See Vol. I, p. 860 and Vol. II, p. 115.)

MORBID GROWTHS OF THE LYMPHATIC GLANDS AND VESSELS

Those of any form are rare in early life. Primary *lymphosarcoma* is occasionally seen in the cervical or in the tracheobronchial glands. It grows rapidly and soon invades the surrounding tissues, this distinguishing it from Hodgkin's disease. Unless relieved by operation the patient dies in a few weeks or months. Cavernous *lymphangioma* of the tongue has been reported as a congenital occurrence producing macroglossia. It is an angiomatous dilatation of the lymphatic vessels. A similar condition is occasionally seen in the lips, mouth, neck, axilla, or behind the knee. A *cystic lymphoma* also occurs, oftenest in the neck. (See Hygroma, Vol. I, p. 691.)

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[1] Quart. Journ. of Med., 1911, IV, 523.

SECTION XI

DISEASES OF THE DUCTLESS GLANDS AND THE INTERNAL SECRETIONS

Although a ductless gland, the spleen and its diseases have been more conveniently discussed in a separate section. Included in the present chapter, with other conditions of which we have a clearer conception, are certain disorders not yet fully understood, among them infantilism and gigantism, which in some cases, at least, would appear to depend upon some disturbance of the internal secretions. Our knowledge of the action of these secretions and of the functions of the internal glands in general is constantly increasing, although as yet comparatively little is known. It is beyond question that their influence upon development and upon many of the functions of the body is very decided. It is not, however, always clear in what way these influences are brought about. Certainly in many cases there is an interrelationship of the different glands, and the action of one is opposed to or increased by that of another. It is consequently difficult to determine upon just what disturbance a certain symptom depends, since apparently one or another gland may be operative in different, although similar, cases. The statements which follow are therefore to be accepted as provisional, and liable to modification as our knowledge of the subject increases.

CHAPTER I

DISEASES OF THE THYROID GLAND

CONGESTION OF THE THYROID GLAND

Congestion may occur under a variety of circumstances. In girls at about the time of the establishment of menstruation there may be observed a congestive enlargement of the thyroid gland which lasts for months. This frequently is a cause of anxiety to the parents, since it suggests goitre. Generally it disappears entirely. Congestion of the thyroid may be seen also in the course of certain diseases, such as affections of the heart and in many of the acute infectious disorders, among them pertussis, scarlet fever, measles, typhoid fever, and diphtheria; a hyperactivity of the glandular function being associated with the congestion. School-epidemics of congestion have been observed. An enlargement which lasts for any considerable time should be suspected, as it may be the beginning of goitre. Temporary congestion may result from hard exercise, too tight collars, or long crying; or in some cases may always develop during sleep. There are no symptoms characteristic of congestion and no treatment is required.

THYROIDITIS

This very uncommon affection may be either primary as the result of of trauma or of some undiscoverable cause; or oftener secondary, occurring in the course of some of the infectious fevers, sepsis, or rheumatism. The symptoms consist of swelling, pain and tenderness in the region of the thyroid gland, with limitation of the movement of the neck and pain on swallowing. The inflammation usually involves one lobe only, or one more than the other. Constitutional symptoms, with fever, are usually present. Generally the cases terminate in resolution after 3 or 4 days of illness, and even in the rarer suppurative cases the prognosis is good, except for the destruction of more or less of the gland, and the symptoms which may result from this. Treatment consists in the employment of cold applications to the neck, and an incision if suppuration takes place.

GOITRE

Etiology.—The disease may be either congenital or acquired; endemic or sporadic. It is not uncommon in children living in districts where goitre is endemic. Demme[1] reported 642 cases coming under observation in the hospital in Berne under the age of 15 years. In 37 of this series the goitre was present at birth or within a few days, and in 96 in all it appeared in the first 2 years of life. He designated 53 of the cases as congenital in origin. In other parts of the world, however, goitre is rarely seen in early life. The frequency increases as puberty is approached. Females are decidedly oftener attacked. Inheritance is a strongly predisposing factor, especially in the congenital cases, while the character of the drinking water is probably an important cause in the acquired endemic cases, as it is in adults.

Pathological Anatomy.—The lesions are similar to those seen in adult life. The involvement of the gland is generally partial except in the congenital cases, and occurs oftenest on the right side or in the isthmus only; and the changes may be either follicular, cystic, colloid, or fibroid; the last two being uncommon in early life. Sometimes the parathyroid glands are involved in the process and enlargement of the thumus gland may also be present.

Symptoms.—A large number of patients with endemic goitre are cretins. Yet cretinism is not a symptom of enlargement of the thyroid gland, but of hypothyroidism; a condition which may result either when all the functionating tissue of the thyroid gland is involved in goitre, or when the gland is absent or atrophied. There may be cretinism without goitre, or goitre without cretinism. The symptoms of goitre are of a mechanical nature, as in adults. In some congenital cases the enlargement has been great enough to interfere with birth. Generally, however, it is of a much less degree. A very moderate enlargement of the thyroid gland may be visible, if there is not much subcutaneous fat. Owing to the high position of the gland in the short neck of infants and the softness of the trachea, even a comparatively small and undiscoverable goitre may readily and rapidly produce severe disturbance. The symptoms consist principally in interference with respiration. The inspiration is prolonged and noisy, and there is constant or paroxysmal dyspnea or hoarseness. The development of a comparatively acute thyroiditis may produce rapid increase in the size of the goitre and very threatening asphyxia. Compression of the veins may also occur and give rise to symptoms such as vertigo and headache. Atelectasis, pulmonary edema, bronchopneumonia, cardiac dilatation, and softening of the tracheal cartilage may occur as complications.

Prognosis.—This depends upon the size and situation of the enlargement. On the whole it is favorable. In very many instances the goitre remains small or diminishes under treatment or spontaneously. The last is true especially of the congenital cases. When, however, pressure does exist, the results of this are liable to be more rapidly serious than in adult life and may even be fatal.

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[1] Gerhardt's Handbuch der Kinderkrankheiten, 1878, III, H. 2, 369.

Diagnosis.—This is readily made. Goitre is to be distinguished from congestion by the persistence, and by the limitation to a part only of the gland, which usually characterizes acquired cases. Other tumors in the neck are possible, but uncommon in children.

Treatment.—Surgical treatment is to be employed only in cases where other means have failed. The continued administration of iodine is generally the best remedy. For this purpose may be employed the syrup of the iodide of iron or small doses of iodide of potassium (½ to 3 grains) (0.032 to 0.194). The possibilities of producing a chronic iodism are not to be forgotten. The administration of thyroid extract has been recommended as a means of giving iodine, but offers no especial advantage. It should certainly be tried if evidences of hypothyroidism are present. Change of climate is often of value, particularly removal to the seashore. The medical treatment will be successful in about 90 per cent. of the cases (Kocher).[1] Recovery is, however, not rapid, and long continuance with the therapy is necessary, especially to prevent the not uncommon relapses. In cases which do not yield to treatment and in which dangerous pressure-symptoms are present, operation is required.

SPORADIC CRETINISM

(Infantile Myxedema; Athyriosis; Hypothyroidism; Myxidiocy; Congenital Myxedema)

Endemic cretinism is confined to certain regions in Europe. It is closely associated with endemic goitre, many of the patients exhibiting goitrous enlargement of the thyroid gland, or being born of parents with this disease. On the other hand, there are often cretins who do not suffer from goitre, and the two conditions are not necessarily combined. The direct cause is hypothyroidism, but the ultimate cause of this is not certainly known. The disease is in its symptoms so closely allied to sporadic cretinism, except for the presence of goitre, that no further description will be given.

Sporadic cretinism was shown by Bourneville and d'Olier[2] to be identical in nature with the myxedema of adult life, to which attention was first called by Gull.[3] It differs from the endemic form in that goitre is not often present and there is usually no hereditary or familial relationship shown. It is not of very common occurrence, but more so than was formerly thought.

Etiology.—A somewhat larger number of females than of males are attacked. The cause is deficiency of the internal secretion of the thyroid gland. This depends oftenest upon absence or atrophy of the organ, or at least an impairment of function as illustrated in the cases with hypertrophy. The degree of cretinism depends upon the degree of hypothyroidism. The disease is usually congenital, even although symptoms do not manifest themselves at once. It is possible that a delay in the appearance of symptoms until the latter part of the 1st year depends in some instances upon the presence of thyroid secretion in the milk of the mother or received through the placenta and effective for some time; or it may be the result of a protective action of the thymus gland.

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[1] Correspbl. f. Schweizer Aerzte, 1898, XXVIII, 545.
[2] Prog. méd., 1880, VIII, 709.
[3] Trans. Clin. Soc. London, 1874, VII, 180.

Cretinism in cases not congenital (*acquired cretinism; hypothyroidism; juvenile myxedema*) may depend upon a lesion of the gland occurring in the course of some acute disease, oftenest an infectious fever. It may also follow the development of sporadic goitre or be the result of a disturbance of function brought about by some unknown cause.

Pathological Anatomy.—The thyroid gland is generally atrophied or absent; yet there are cases in which cretinism has existed with an apparently normal thyroid. The liver and spleen are often enlarged. The skin and mucous membranes, as in the myxedema of adults, are thickened by myxedematous changes. In the long bones the centres of ossification are very late in appearing, and the entire process of osseous development is very slow, both in the epiphyses and in the diaphyses (Fig. 407); and the same is true of the bones of the cranium. The condition is thus just the opposite of rachitis, in which there is an excess of growth in the cartilaginous tissue. There is often present a hyperplasia of the hypophysis cerebri, the pineal gland and the thymus gland.

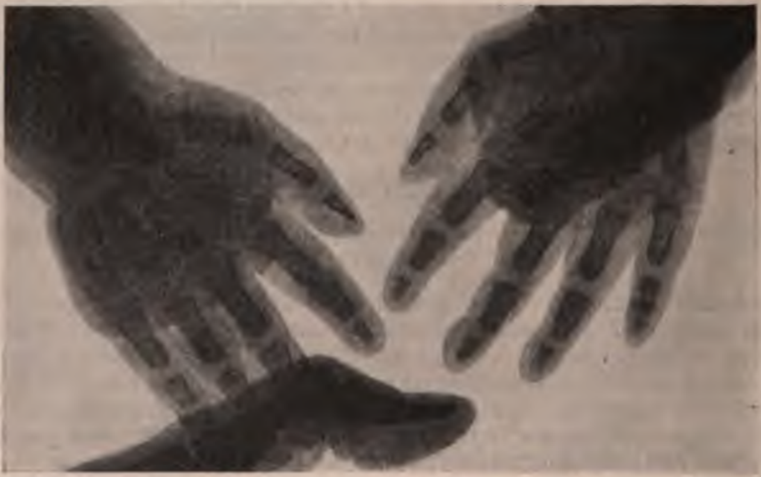

FIG. 407.—CRETINISM, RADIOGRAPH OF THE HANDS.

Showing absence of the epiphyses. (*Judson and Bradley, Arch. of Pediat.*, 1908, *XXV*, 525.)

Symptoms.—The various classifications which have been employed;—congenital myxedema; cretinoid; myxedematous idiocy; infantile myxedema; juvenile myxedema; hypothyroidism, and the like—designate merely differences in the degree of thyroid insufficiency and the time at which this develops. The symptoms are generally not observable before 6 months of age, and in the mild cases not until the child is 2 or 3 years old. Taking as a type the average case of congenital cretinism, the appearances are extremely characteristic. The body as a whole is much stunted in growth, thick-set and with short legs; the child of 5 years seeming at first sight like a baby of little over a year, and an adult being no longer than, if as along as, a child of 4 or 5 years of age. The head appears large, and the fontanelle is widely open much after the normal time for closing, or even into later childhood. The hair of the scalp is coarse and scanty; the forehead low; the eyes far apart; the palpebral fissure narrow; the eyelids swollen; the bridge of the nose broad and flat and the alæ thick. The tongue seems much too large for the mouth, being thick and broad and often protruded constantly or much of the time; the lips are thick, and the teeth are late in appearing and frequently decay early. The face appears bloated and sallow, often exhibits deep creases in the forehead and elsewhere, and the whole expression is blank, dull and apathetic. The neck is short and thick; the thyroid gland cannot be felt, and there are often fatty deposits above the clavicles and sometimes also between the scapulæ or in the arm-pits. There is no alteration in the shape of the chest, but the abdomen is prominent and umbilical hernia is common. The hands are broad, with short, stumpy fingers; the toes short; and the hands and feet often cold and cyanotic. The legs and arms are short and often crooked. The skin of the body is harsh and dry, thickened, and in creases as though too abundant. It appears at first to be edematous, but does not pit on pressure. There is but little perspiration; the subcutaneous fat is abundant; meta-

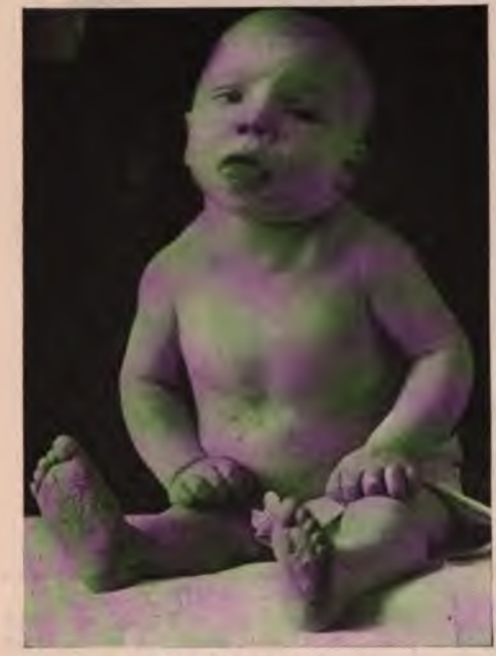

FIG. 408.—CRETINISM.

Male, aged 6 years. Patient in the Children's Hospital of Philadelphia. Protrusion of tongue noticed in the 2d month. Child cannot talk, understands but little, and is very unobservant. Tongue very large, hands short and pudgy. Improved decidedly under treatment.

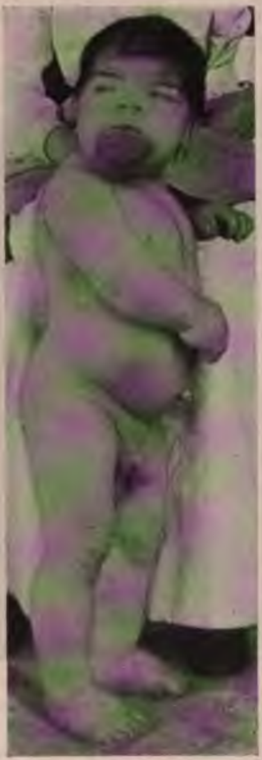

FIG. 409.—CRETINISM.

Patient, aged 14 years in the Children's Ward of the Hospital of the University of Pennsylvania.

bolic activity is low; the body-temperature subnormal, and the patient easily chilled. Constipation and anemia are common. Even as age advances there is no sexual development. The attaining of the bodily power is slow; muscular movements being few and made slowly, and standing and walking not being learned until very late, and in the worst cases not at all. The intelligence, too, is extremely backward, and the acquiring of a hoarse speech is late or absent altogether. The disposition is apathetic, placid and good-natured, and much of the time is spent in sleep (Figs. 408, 409).

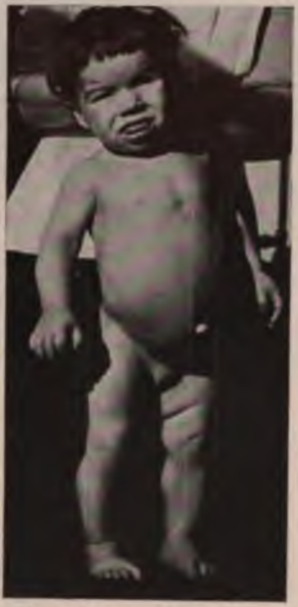

FIG. 410.—CRETINISM.

Patient, aged 4 years in the Children's Hospital of Philadelphia. Takes little interest in surroundings and cannot walk or talk.

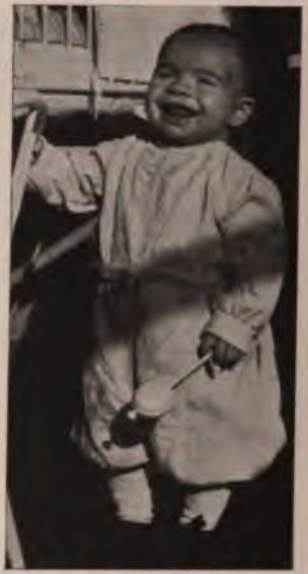

FIG. 411.—CRETINISM, SAME CASE AS IN FIG. 410.

Photograph taken 25 days later. Lost 3 pounds (1361 gm.), is walking by support of the furniture, feeding herself, says a number of words plainly, alert to surroundings.

Great variation is seen in the degree to which these symptoms develop. In the mildest cases (*cretinoid; hypothyroidism; incomplete myxedema*) in which the function of the thyroid gland is present to some extent, the growth of the body is stunted usually to but a moderate degree and the mental state but slightly altered; although the expression generally characteristic of the cretin is readily discoverable in a modified form. Such subjects are able eventually to learn to do certain kinds of work. They are instances of incomplete hypothyroidism. The worst cases of cretinism, on the other hand, begin to exhibit the evidences of the disease even sometimes in the early months of life; and the patient in adult life retains the bodily and mental characteristics of the infant, and is a complete idiot. Still other cases enter upon life in a normal state and continue so for perhaps 4 or 5 years; and after this, as a result of some acute disease, or without discoverable reason, begin to develop symptoms. Bodily growth is then largely arrested and the intellectual power which had been attained retrogrades to a considerable extent, the degree depending upon the age when the disease began. The facial expression becomes that of cretinism and the characteristics are in general the same, although modified by the time of the development of the disease. A few cases of *operative myxedema* occurring in children are on record, dependent upon removal of the thyroid gland.

Course and Prognosis.—The course is generally slow, and especially so in the atypical instances with but slight cretinoid changes. In such there is no curtailment of the duration of life. Of the average cases, however, very many die in childhood, and the majority do not reach adult life; some complication being generally the direct cause of death. The prognosis in patients receiving treatment depends upon the age at which this is commenced and the severity of the symptoms. In some the improvement is really wonderful (Figs. 410–413); in others it is but partial, and in some none at all can be obtained. In no case can we expect a complete removal of all evidences of hypothyroidism.

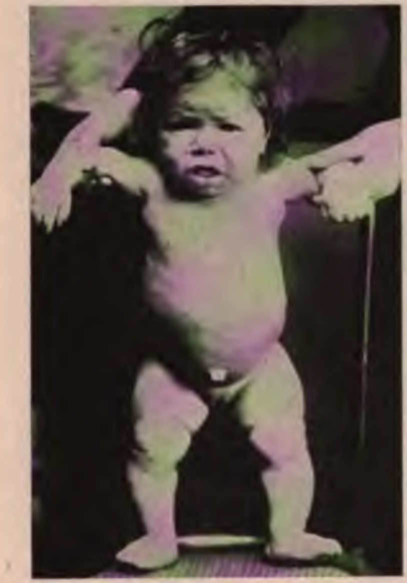

FIG. 412.—CRETINISM.

Patient, aged 2 years and 1 month. Before treatment. *Courtesy of Dr. A. H. Davisson.*

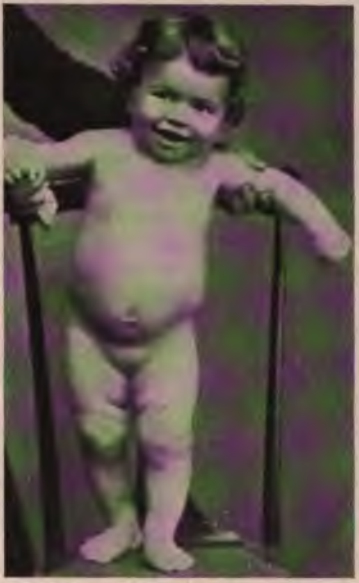

FIG. 413.—CRETINISM.

Same case as in Fig. 412, now aged 3 years and 2 months. Condition after 13 months of treatment with thyroid extract.

Diagnosis.—In the typical cases the diagnosis can hardly fail of being made. The stunted growth; the vacant expression of face; the large, thick, protruding tongue; the coarse hair, and the fatty tumors are characteristic. Early in the course and especially in infancy mistakes are readily made. The condition causing most confusion is *Mongolian idiocy.* In both disorders the tongue is protruded, the palpebral fissure is narrow and the intelligence is below normal. The tongue of the Mongolian idiot, however, is not really enlarged; the eyes often have the orient slant; the child is restless rather than apathetic; and the hands have not the broad, squat-form of the cretin, and often exhibit a characteristic incurving of the little finger. Yet I have seen a number of instances in which the early diagnosis was most uncertain and could be but provisional. *Achondroplasia* produces shortness of the extremities combined with a large head and a flattening of the bridge of the nose; but in other respects there is no resemblance and the facial expression and mental state are entirely different. *Rachitis* may show stunted growth, large open fontanelles, delayed dentition, protuberant abdomen, curvature of the extremities and inability to stand or walk; but in the facies, and in other respects, it bears to cretinism no resemblance whatever. In rachitis, too, there is characteristic enlargement of the epiphyseal junctions and free sweating. *Infantilism* with a large amount of subcutaneous fat bears a resemblance to cretinism chiefly in the dwarfism present; although in some cases there may be retardation of mental growth and failure of development of the genitals. There is, however, in most classes of the condition an absence of the cretinoid facies and the skin is not myxedematous. Some instances of infantilism probably depend upon hypothyroidism and may well be regarded as atypical cases of cretinism. (See Infantilism, p. 533.)

Treatment.—Fortunately there is found in the administration of the thyroid gland of other animals a specific which is of avail in many cases. Treatment at first consisted in the transplantation of the gland. Extracts have been given hypodermically, and the freshly prepared gland or a glycerine extract from it administered by the mouth, as well as various commercial derivatives. At present the administration by the mouth of the dried, powdered gland, denominated "extract," is usually adopted. It is important that a reliable preparation be obtained. The earlier treatment is begun, the greater the chance of benefit; and the improvement seen is sometimes remarkable, even when the patient is several years of age before treatment was commenced. Especially in infancy the remedy must be administered with caution, inasmuch as a thyroid intoxication is readily produced by over-dosage. Among the symptoms of this are increasing debility, rapid loss of flesh, fever, rapid cardiac action, nausea, restlessness, perspiration, faintness, sleeplessness, and other constitutional disturbances. The initial dose should not be over ¼ grain (0.016) twice daily, and this may be steadily increased to a dosage of 5 grains (0.32), if no unfavorable symptoms appear. In cases responding to the treatment the first evidences of improvement, seen in the course of a few weeks, are a disappearance of the subnormal temperature, a moderate loss of weight due to the removal of the myxedematous infiltration, and a diminution in the size of the tongue. The teeth make their way through the gums; increase in length begins; the child commences to walk; the idiotic expression of the face diminishes; the character of the hair changes; constipation ceases, and the intellectual power improves. Physical development, however, generally takes place before that of the mind, and may be all that is gained. It is imperative that treatment be continued constantly during life, although generally in lessened dosage, and with occasional intermissions of a few days or a week, if the dose appears to have been too large. If medication is suspended, relapse will certainly follow.

Apart from the specific treatment certain general measures are important. Inasmuch as the circulation seems often poor, care must be taken to keep the patient in an even temperature. A warm climate is to be preferred in the winter season. It has been observed that the condition often begins to grow worse after weaning, and it is the general experience that although the diet should be nourishing, the amount of meat ingested should be diminished. Milk, amylaceous food and fresh vegetables should constitute a large part of the dietary. Sugar is tolerated by cretins in a quantity which would produce alimentary glycosuria in a healthy subject.

EXOPHTHALMIC GOITRE

(Graves' Disease; Basedow's Disease; Hyperthyroidism)

Etiology.—In its nature this disease is the reverse of cretinism, there being present a hyperthyroidism. Graves[1] in 1835 was the first to describe it fully, although cases had been reported by earlier writers. The disease is rare in childhood, Barret[2] in 1901 being able to collect but 39 cases under 15 years of age, 3 of the patients being under 5 years of age; 11 from 5 to 10 years old; and 32 of the 39 cases between 8 and 15 years, the youngest patient being 2½ years of age. Yet the malady has been known to appear congenitally (White),[3] and Klaus[4] reported an instance in an infant of 9 months. The influence of inheritance may be decided, the parents having had simple or exophthalmic goitre, or having been neuropathic or alcoholic. Gurney[5] in an analysis of 93 cases of all ages found hereditary or familial influence in 10.7 per cent. Females are much oftener attacked. The development of the condition may have been preceded by shock, fright, blows on the head, or the occurrence of some infectious disease; or exophthalmic goitre may exist as a complication of a simple goitre.

Pathological Anatomy.—The only characteristic lesion is the enlargement of the gland, dependent upon a true hyperplasia of the adenomatous tissue. The thymus is usually found persistent or enlarged and the spleen occasionally enlarged as well. The increase in size of the thymus has led to the belief that a disturbance of the function of this gland is active in producing the disease, in combination with that of the thyroid (Matti).[6] No characteristic lesion of the sympathetic nerves appears to exist, although the disease is essentiallly in its symptoms a disturbance of this system.

Symptoms.—These do not differ very materially from those observed in adult life. The cardinal symptoms most frequently present in children are tachycardia, which is often the first seen; enlargement of and pulsation in the thyroid gland; and exophthalmos. Tremor is less common than in adults, or may be replaced by coarser movements suggesting chorea; and the exophthalmos, although usually present, may often reach the degree only of a staring expression. Among other symptoms observed are enlargement of the heart; cardiac murmurs; occasional vomiting or diarrhea; dyspnea; anemia, debility and emaciation; headache; retraction (Stellwag's sign) or lagging (von Graefe's sign) of the upper lids; forcible pulsation in the neck and in the peripheral vessels; elevation of temperature; and perspiration, flushing, pigmentation or other cutaneous manifestations. The psychic state is always disturbed, although to a less extent than in adults. There is irritability, great restlessness and disturbed sleep, often hyperexcitability, sometimes depression.

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[1] London Med. and Surg. Journ., 1835, VII, 513.
[2] Thèse de Paris, 1901.
[3] Proc. Royal Soc. of Med., 1912, V, Obstet. Sect., 247.
[4] Prag. med. Wochenschr., 1914, XXXIX, 515.
[5] Brit. Med. Journ., 1915, I, 924.
[6] Berl. klin. Woch., 1914, LI, 1310.

This description applies to the more fully developed cases. Not all the symptoms are always present, and any one of the three chief manifestations may be the first to appear and perhaps be the only one for a considerable time. The term *hyperthyroidism* is often applied to the "incomplete" or "abortive" cases of exophthalmic goitre, more frequently observed than the typical ones. In these there is a perversion of function of the thyroid gland with but little increase in the size; a slight staring expression of the eyes; increased rapidity of the heart's action; irritability, restlessness and other nervous symptoms; and often moderate anemia and loss of weight.

Course and Prognosis.—Although the disease may be either acute or chronic in its course, in early life the onset is oftenest sudden and the development of symptoms rapid. The complete picture may be attained after a few weeks or even a few days. The duration on the whole is less than in adult life; complete recovery occasionally taking place in the course of a few weeks; or, even when delayed, occurring finally in over one-half the cases. In other instances great improvement takes place; but slight enlargement of the thyroid gland, a disposition to tachycardia, or a tendency to relapse remains. In the more frequent chronic cases there is a gradual development of anemia, emaciation, and debility, owing to the increased degree of metabolic change going on; pigmentation; a ready exacerbation of symptoms; and the disease may last from 6 months to several years. A few cases are on record in which a myxedematous condition had developed as a sequel to a final atrophy of the thyroid gland (Baldwin.)[1] The prognosis for complete or partial recovery is, therefore, good on the whole, although always there is the great tendency to recurrence. Death from the disease itself is exceptional in early life. The mortality may be estimated as not equalling 10 per cent. at this period. Only 2 of Barret's series of 39 cases terminated fatally.

Diagnosis.—In the well-marked cases this is easy; but those with incomplete development of the clinical picture present many difficulties. In such frequently only a tentative diagnosis can be made. The combination of a nervous condition with a rapid cardiac action without organic lesion is always suspicious. Only the development of exophthalmos with enlargement of the thyroid gland can make the diagnosis sure. The various symptoms of hyperthyroidism described, often combined with a slight enlargement of the thyroid gland, and occurring not very infrequently as a temporary condition in children exhibiting rapid growth, are to be considered as evidences of incomplete exophthalmic goitre.

Treatment.—Many measures have been recommended. Among those offering the best hope of cure are rest in bed in the open air; avoidance of all exertion and excitement; warm baths or packs; galvanization of the thyroid gland and of the sympathetic nerve in the neck; cold compresses over the heart; and the administration of such drugs as strophanthus, belladonna, the bromides, arsenic, and cod-liver oil. The use of internal remedies is, however, of minor importance. Specific treatment has been employed with some success. One method consists in the internal administration of the serum of dethyroidized sheep; while Rogers and Beebe[2] have employed the serum of animals injected with the extract of human thyroids. The favorable course in children renders this specific treatment as well as all surgical procedures usually unnecessary.

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[1] Lancet, 1895, I, 445.

[2] Journ. Amer. Med. Assoc., 1906, XLVI, 484; 487; Arch. Intern. Med., 1909, II, 297.

OTHER DISORDERS OF THE THYROID GLAND

Tumors of any sort are of very exceptional occurrence. Primary sarcoma has been reported in a few instances, developing in a goitre already present, and carcinoma even less often. Secondary growths are somewhat more frequent. Enchondroma has been seen. Aberrant thyroid tissue in the course of the neck may develop into tumor-masses or cysts. This may be observed, for instance, in the tumors of the "lingual thyroid" attached to the hyoid bone or situated in the deep muscles of the tongue.

Tuberculosis may affect the thyroid gland secondarily in advanced cases of this disease elsewhere; and hereditary **syphilis** may rarely produce a goitrous enlargement, perhaps containing gummata. **Cysts** may form in connection with other disease of the organ. These occur in goitre; but are also the result in some cases of a failure in development.

DISEASES OF THE PARATHYROID GLANDS

These glands, from 1 to 4 in number, are attached to the lateral lobes of the thyroid gland on their posterior aspect. They appear in some way to be necessary to life and their accidental injury in operation upon the thyroid gland may be followed by tetany. If they are completely removed, death will take place. Exactly what their influence may be in the production of tetany in children has not been satisfactorily determined. It is possible that they may be active through their influence upon calcium metabolism. (See Tetany, p. 251.) In at least some cases of this disease autopsy has shown hemorrhage in these glands, while in others no lesion has been discovered.

CHAPTER II

DISEASES OF THE THYMUS GLAND

Both the physiological function and the diseases of the thymus gland are as yet but little understood, although the subject has been very extensively studied. Even its anatomy is not definitely known, especially as regards its size in health. Its dimensions and weight appear to vary so greatly within normal limits, that the determination at autopsy that the condition is abnormal can be made only when the variations from the average are extreme. Equally disputed is the power of the thymus gland to produce symptoms, either mechanically or by a disturbance of its internal secretion. (See Thymus Gland, Vol. I, p. 62.) It is, however, now generally believed that the thymus gland possesses an internal secretion and that this is a factor, either alone or in combination with that of other ductless glands, in affecting the economy in ways not clearly comprehended. Among other properties the gland has been believed to influence the development of the bones, as in rickets; to be by the production of a toxin the cause of sudden death in the lymphatic diathesis; to be a factor in the causation of exophthalmic goitre; to increase the coagulability of the blood; to be an important regulator of blood-pressure; to produce symptoms suggesting tetany; to possess some influence upon the development of the nervous system and the sexual organs; to be of importance in the production of chlorosis and the primary myopathies. It is at least certain that much further investigation is required.

ABSENCE AND ATROPHY OF THE THYMUS GLAND

In rare cases there may be congenital *absence* of the thymus gland. This is generally associated with other malformations of some sort, especially anencephalus, or with defective mentality. A few uncomplicated cases are on record. *Atrophy* of the thymus gland is a natural occurrence as adult life is reached; but it may be seen also under a number of other conditions. It has been observed in the new born (Durante),[1] and is found often in the case of infants dying in a state of malnutrition. To this especial attention has been directed by Stokes, Ruhräh and Rohrer[2] among others. The assumption has sometimes been made that this thymic atrophy was the cause of the general wasting; but it is much more probable that it is merely a part of the latter. To atrophy and a consequent loss of function has been ascribed the development of rickets and other conditions. There are, in fact, no special symptoms connected with atrophy of the gland, and the results produced by experimental removal of it in young animals have been inconclusive and to some extent contradictory; although often there appears to follow some interference with metabolism and nutrition.

ENLARGEMENT OF THE THYMUS GLAND

Etiology and Pathology.—A *persistence of the thymus gland* during adolescence and into adult life is sometimes observed. It appears to be unproductive of symptoms and without clinical interest. A true *hyperplasia* in early life is of common occurrence, and not infrequently seen even after this period. The gland may sometimes be so much enlarged that it nearly covers the heart and projects well beyond the internal borders of the lungs, and occasionally weighs as much as 30 or 40 grams (1.05 or1.4 oz.) or more. The subject of the causes and clinical manifestations of hyperplasia has been, and still is, a matter of much discussion. As already stated, the range of size of the gland within normal limits is so great that it is difficult to determine the exact definition of what is and what is not hypertrophy. (See Thymus Gland, Vol. I, p. 62.) It is only, therefore, in cases where the size and weight of the organ is exceptionally great that we can with certainty speak of hypertrophy (Fig. 414). This hypertrophy is generally the result of a simple hyperplasia, but it may depend upon a thymitis or exceptionally upon hemorrhage or edema. Its cause is far from being understood. That the condition is a frequent one in lymphatism has already been pointed out (Vol. I, p. 632); but a very large thymus gland may occur in children who have no symptoms of this disorder. It has been found in combination with a number of morbid conditions, among them exophthalmic goitre, acromegaly, epilepsy, leukemia and Hodgkin's disease. There seems to be an intimate association of the thymus with the thyroid, spleen, pituitary body, adrenals and other internal glands. Whether the thymic hyperplasia is a compensatory process in diseases of these organs, or whether there is an accompanying disturbance of the thymic function which increases the symptoms attributed to disorders of the other glands is a matter for further study.

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[1] Comptes. rend. soc. biol., 1896, XLVIII, 285.

[2] Amer. Journ. Med. Sci., 1902, CXXIV, 847.

Symptoms.—The position and size of the thymus may be determined by physical examination. The studies in percussion, especially by Blumenreich,[1] on subjects up to 6 years of age, show that the absolute dullness which may be assumed as a normal average one, occupies an inverted triangle in the region of the upper part of the sternum, extending transversely from the right sternoclavicular articulation to a point slightly beyond the articulation on the left side, and with its apex in the central line of the sternum between the articulations of the 2d ribs or sometimes slightly lower, the triangle being irregularly shaped, with the larger portion to the left (Fig. 415). This leaves an area of resonance above the upper border of the cardiac dullness at the 3d rib. A percussion dullness which extends 1 cm. (0.4 inch) beyond these limits indicates that the thymus gland is enlarged. In some children the normal thymus gland may be palpable in the episternal notch. Radiography, too, as urged by Hochsinger[2] may be serviceably employed. Yet results obtained by any method of physical examination must be accepted with caution, owing to the normal range in the size of the gland referred to,

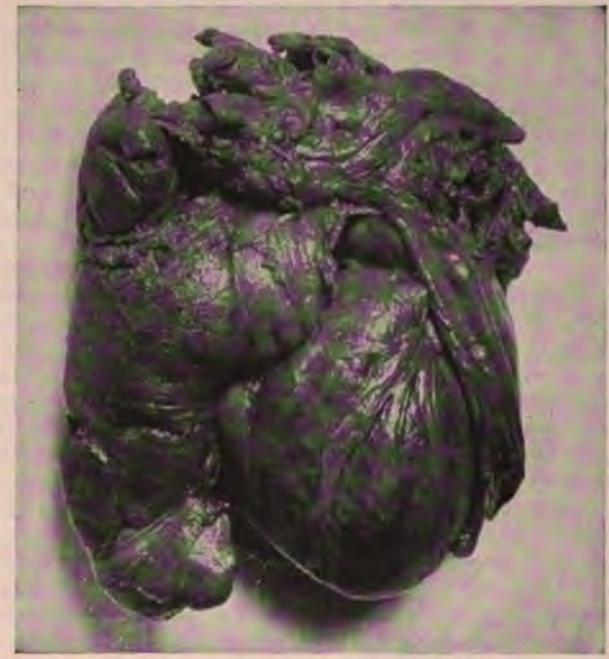

FIG. 414.—ENLARGED THYMUS GLAND.

Infant of 3½ months dying after a few hours illness. Rapid respiration and cyanosis, but no symptoms of obstruction. Autopsy showed the thymus gland measuring 7 cm. (2.76 in.) in length, 3½ cm. (1.38 in breadth and 2 cm. (0.78 in.) in thickness. *Courtesy of Dr. Eleanor Jones.* (*See also, Arch. of Pediat.*, 1903, *XX*, 596.)

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[1] Virchow's Archiv., 1900, CLX, 35.

[2] Wien med. Woch., 1903, LIII, 2106.

and especially as it is possible for enlarged tracheobronchial lymphatic glands to present similar signs. Benjamin and Gött[1] have shown, too, that the *x*-ray shadow by no means corresponds with the thymus gland as seen at autopsy. They believe the shadow is produced by the superior vena cava.

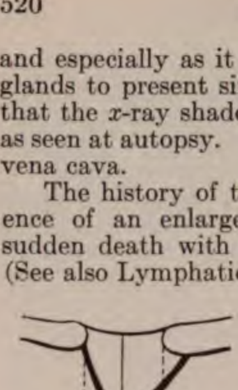

Fig. 415.—Diagram Showing Normal Thymus Dulness.

(*Blumenreich, Virchow's Archives*, 1900, *CLX*, 34.)

The history of the symptoms which have been assigned to the presence of an enlarged thymus gland, and especially the connection of sudden death with thymic hypertrophy, is a long and interesting one. (See also Lymphatic Diathesis, Vol. I, p. 632.) In brief it may be stated that Plater[2] in 1614 noted the frequent presence of thymic enlargement in cases dying suddenly; and Kopp[3] in 1830 maintained the existence of a "thymic asthma" dependent upon the pressure of such a gland upon the trachea. This view held sway until banished by the very classical investigations of Friedleben,[4] which led to the conclusion that there was no connection whatever between enlargement of the thymus gland and asthma or sudden death. A reversion of sentiment in favor of compression occurred under the influence of Grawitz;[5] but only a short time later Paltauf[6] advanced the theory of the existence of what he called the "status lymphaticus," which has been since much discussed and largely adopted. According to this view, enlargement of the thymus gland is but a part of the general involvement of the lymphatic system occurring in the lymphatic diathesis, or the status lymphaticus; and the symptoms do not depend upon the thymus itself, or certainly not upon any enlargement of this producing mechanical interference, but upon a neurosis. Cases, however, continued to be reported indicating the possibility of pressure by the thymus gland, and the production of corresponding pressure-symptoms, or even of death brought about in this way. Many of these do not stand the test of scientific analysis, but certain of them are beyond question, and it would appear to be indisputable that the so-called "thymus-death," as well as other symptoms connected with thymic hyperplasia, may be of different sorts and produced in different ways. These may be classified as follows:

1. *Pressure Upon the Trachea.*—There is no doubt that such pressure may occur and that symptoms are produced by it, although not nearly so often as supposed by some. There are numerous reported cases in which sudden death has taken place and an autopsy has shown an enlargement of the thymus gland; but no sufficient proof is offered in most of these that the enlargement had by pressure been the cause of the symptoms. In a collection of cases made some time ago[7] I could find only in the neighborhood of 20 in which autopsy had revealed undoubted compression, but even then it remained doubtful whether the compression had been the cause of death. In addition to these there were, of far more value, 10 instances in which operative interference had given

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[1] Jahrb. f. Kinderh., 1912, LXXV, 367.
[2] Observat. in homin. affect. pleurisque, 1614, lib. III, 172.
[3] Denkwürdikeiten, 1830, I.
[4] Die Physiologie der Thymusdrüse, 1858.
[5] Deutsche med. Wochenschr., 1888, XIV, 429.
[6] Wien. klin. Wochenschr., 1889, No. 46: 1890, No. 9.
[7] New York Med. Journ., 1909, Sept. 4.

partial or complete relief from the symptoms; and 1 in which the employment of the *x*-ray had had the same good results. I have myself had 1 case in which *x*-ray treatment gave complete relief, radiography meanwhile showing disappearance of the thymic hypertrophy; and the number of recorded cases of relief of symptoms by thymectomy or by the employment of the *x*-ray has increased very decidedly since the report referred to. Parker[1] collected 50 instances of thymectomy, usually with complete relief, although there were 17 fatal cases. Klose[2] at a later date made the number 58. But in all such instances the disease is of a chronic nature, the symptoms consisting of more or less persistent tracheal stenosis, either always present with exacerbations, or frequently coming on in repeated paroxysms, the condition lasting for weeks or months. Cases of *congenital laryngeal stridor* have been claimed by Hochsinger[3] to be the result of pressure of an enlarged thymus gland. Some of these are probably due to thymic enlargement; others certainly depend upon different causes. (See p. 26.)

2. *Pressure Upon Other Organs.*—Theories have been advanced that symptoms may result from pressure of the thymus gland upon the large vessels within the thorax, upon the nerves, or upon the heart itself. These have not obtained general acceptance and lack sufficient anatomical confirmation.

3. *Symptoms Other Than Those of Pressure Attributed to Thymic Hyperplasia.*—In this category are to be placed the not infrequent cases of sudden death, sometimes entirely without warning, sometimes preceded by earlier threatening attacks. These are the deaths more properly assigned to lymphatism (Vol. I, pp. 216 and 632). Many of the cases appear to be instances of laryngospasm, but are more probably death through sudden cardiac failure. This condition is a neurosis, in all likelihood dependent upon an autointoxication, the origin of which is not certainly known. Whether the thymus gland has anything directly to do with it is doubtful. Although a disturbance of the function of the thymus, and an intoxication by its secretion, is conceivable, as Escherich[4] believed, this has never been proven. Švehla,[5] Basch[6] and others have claimed decided, although varying, effects in animals from the injection of an extract of the thymus gland, and a condition of hyperthymization has been spoken of. On the other hand, injections made by Fischl[7] were entirely without serious specific action. Hammar[8] in a very careful microscopical study of the thymus glands from 13 cases of so-called thymus death in children found no characteristic morphological changes, no marked variations in size, and no ground for assuming the action of either an increased or a diminished function.

Prognosis and Treatment.—The prognosis of true thymic asthma, dependent upon pressure upon the trachea, is on the whole good. There is a tendency to recover in the milder cases as the child grows older; and in the severer ones there is usually time for successful treatment to be instituted. This may consist in the employment of the *x*-ray, if the case is not urgent. In other instances of a more threatening nature a thymectomy, or the lifting and anchoring of the thymus gland by suturing, has been successfully practised. It is probable that the x-ray is not entirely without danger, and thymectomy is a distinctly serious operation. Nevertheless, in the condition of pressure in which any such treatment is indicated, the disease is a dangerous one and the risk must be taken. The treatment of thymic enlargement in the status lymphaticus, without distinct pressure symptoms, is of an entirely different nature, and has already been described (Vol. I, p. 634). No therapeutic measures directed against the thymus are indicated here, because its enlargement is a symptom and not the cause of the condition.

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[1] Amer. Jour. Dis. Child., 1913, V, 89.

[2] Therapeut. Monatsh., 1915, XXIX, 6.

[3] *Loc. cit.*

[4] Berliner klin. Wochenschr., 1896, XXXIII, 645.

[5] Wiener med. Blätter, 1896, XIX, 723.

[6] Wien. klin. Wochenschr., 1905, XVI; 893.

[7] Jahrb. f. Kinderh., 1914, LXXIX, 385; 589.

[8] Zeitschr f. Kinderh. Orig., 1916, XIII, 153.

Diagnosis.—The recognition of enlargement of the thymus gland when no evidences of pressure are present can be made only provisionally through the results obtained by percussion and x-ray examination. When pressure-symptoms arise the same difficulty attends the differentiation of hypertrophied thymus from enlarged tracheobronchial glands. The latter affection can produce the same physical signs, and can be distinguished only by other evidences of the presence of this disorder. When interference with respiration is present, thymic enlargement can be diagnosticated from congenital laryngeal stridor with difficulty. The relief which may attend intubation in laryngeal affections aids in differentiating. Death due to mechanical action of the thymus always has a certain element of chronicity about the symptoms, with the previous persistence of some degree of stenosis.

OTHER DISORDERS OF THE THYMUS GLAND

Acute thymitis is a very rare affection, the result oftenest of pyemia. Some of the reported cases of pus-formation were probably only instances of accumulation of the normal secretion. General **tuberculosis** of the body may exhibit miliary tubercles or caseous foci in the thymus gland; and **syphilis** may produce gummata there, or often a small-celled infiltration with consecutive fibrosis. The so-called "Dubois'[1] abscesses" are probably only cystic degeneration of the Hassall bodies dependent upon hereditary syphilis. It is doubtful whether **hemorrhage** into the gland may be found in asphyxiated new-born infants, or in diseases of the hemorrhagic diathesis. **Tumors** of the thymus, either primary or secondary, are unusual. Leukemia may involve the gland; and lymphoma, lymphosarcoma, carcinoma, and teratoma have been recorded.

CHAPTER III

DISEASES OF THE PITUITARY, PINEAL AND SUPRARENAL GLANDS

DISEASES OF THE PITUITARY GLAND

The pituitary body is a complicated organ, the several parts of which probably possess different properties. The anterior lobe influences growth and development; the posterior affects metabolism of the fats and carbohydrates and controls the secretion of urine. The exact function of the gland and that of its individual parts needs still further study, but it is certain that a disturbance of these may produce disease, both by an excess of its internal secretion and by an insufficient amount of this.

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[1] Gaz. méd. de Paris, ser. 3, 1850, V, 392.

ACROMEGALY; GIGANTISM

Acromegaly is regarded as dependent upon an excess of secretion in the anterior lobe of the pituitary body. It is probably unknown before the age of puberty and very rare even at that time, and demands, therefore, no further consideration here. Reference is made to it because there is reason to believe, as suggested by Brissaud,[1] that **gigantism** in some instances is practically the same disease and due to the same cause; the difference being that acromegaly begins after ossification is complete and can produce no increase of the length of the individual, while an excess of pituitary secretion occurring in early life causes a delay in ossification and a consequent increase in the size of the skeleton. In both conditions there is almost always found an involvement of the pituitary gland by tumor, oftenest fibroma or myxoma, or in other ways.

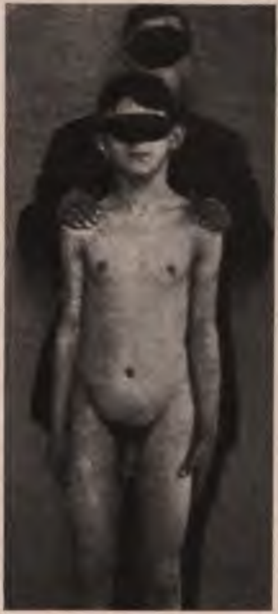

FIG. 416.—PINEAL PRECOCITY.

Patient, aged 7½ years, with father. Height, 145 cm. (57.09 in.), weight, 35.2 kg. *of* (77.58 lbs.) (*Horrax, Arch. Intern. Med.*, 1916, May 15, 631.)

A condition of **hypopituitarism** may produce a form of infantilism (*Fröhlich's Syndrome*), and will be discussed under that topic (p. 533). There may eventually be the occurrence of hypopituitarism as a sequel to hyperpituitarism; or the symptoms may be combined in such a way that the title "dyspituitarism" is well employed (Cushing).[2] There is evidently a close relationship between the function of the pituitary body and that of other glands of internal secretion, such as the thyroid and pineal glands, the suprarenal bodies and the genital glands.

DISEASES OF THE PINEAL GLAND

The function of the pineal gland appears to some degree to be the opposite of that of the pituitary body. Disturbance of the latter may produce failure of development of the sexual organs, while that of the former causes an early overgrowth of these. Lesions of the pineal gland consist almost exclusively of tumors, often teratoma. In some way the gland exercises an influence upon nutrition, in association with other glands possessing an internal secretion. Bailey and Jelliffe[3] collected 60 recorded cases including 1 of their own, nearly half of them being in children and adolescents. Apart from the **symptoms** of cerebral tumors in general, including in a number of cases hydrocephalus, unusual drowsiness, and those indicative of a lesion of the corpora quadrigemina, there are in some instances interesting trophic disturbances. These are seen in children only, since involution of the pineal gland begins to take place at puberty. There are, namely, decided alterations of the sexual and other somatic characters (Fig. 416). These may antedate or follow the general symptoms of cerebral tumor. The syndrome is much oftenest seen in boys between the age of

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[1] Bull. soc. des hôp., 1896, XIII, 3s. 443.
[2] Journ. Amer. Med. Assoc., 1914, LXIII, 1515.
[3] Arch. Intern. Med., 1911, VIII, 851.

4 and 8 years, and consists in abnormal development of the body, an early growth of hair on the pubis and sometimes elsewhere, as on the face; obesity; a marked overgrowth of the penis, and sometimes of the testicles and breasts; and in some cases alteration of the voice. The mental condition is unaffected, except in the later stages as the result of the intracranial pressure. Sometimes there is mental precocity. The published cases of pineal disorder with the characteristic trophic disturbances have been reviewed by Morse.[1]

DISEASES OF THE SUPRARENAL GLANDS

There would appear to be a close interrelationship of the suprarenal bodies and the glands of generation, since both arise from the same fetal organ, the Wolffian body; and yet doubt is thrown on this by the very great difference in structure and in function between the medullary and the cortical portions of the suprarenal glands, suggesting that perhaps they belong to entirely different systems. There is also beyond question some relationship between the functions of the suprarenal bodies and those of some other internal glands, as the thyroid, thymus and pituitary. The secretion of the medullary portion, adrenaline or epinephrine, among other properties controls blood-pressure and influences the metabolism of the skin. That of the cortex is little understood, but certainly has some power over general growth and the sexual characteristics, a hypersecretion producing precocious sexual development, and a hyposecretion giving rise to infantilism.

ADDISON'S DISEASE

Etiology.—This affection, uncommon at any age, is still more so in childhood. Monti[2] in 291 collected cases found but 12 under 15 years of age, the majority of these developing between 12 and 14 years. The youngest was in a child of 3 years. A considerable number have been since added, nearly all at about puberty. Felberbaum and Fruchthandler[3] have collected 25 recorded cases under 14 years, including 1 of their own, and Chemin[4] 55 up to the age of 16 years. A familial tendency has been sometimes observed, as in the instance reported by Fleming and Miller[5] of a mother and 4 children with the disease, and by Croom[6] of another family with 3 children affected. In neither group, however, were there any autopsies.

Pathological Anatomy.—The lesions found in early life have consisted of tuberculous changes in nearly all instances. The process may be extensive, practically destroying both suprarenal glands, or be limited to tuberculous foci in one or both of them. Tuberculosis of other parts of the body may be present. In a small number of cases of Addison's disease, in place of tuberculosis there has been found atrophy or the presence of morbid growths. In some instances fatty degeneration and atrophy have been discovered in the semilunar ganglion and solar plexus.

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[1] Arch. of Ped., 1913, XXX, 179.
[2] Kinderheilkunde, 1903, III, 567.
[3] New York Med. Journ., 1907, LXXXVI, 256.
[4] Thèse de Paris, 1910.
[5] Brit. Med. Journ., 1900, I, 1014.
[6] Lancet, 1909, 1, 603.

Symptoms.—These are entirely similar to those seen in adult life. Although at times acute, the onset is generally slow and insidious. The early general symptoms consist of an increasing debility, a weak action of the heart, vomiting, diarrhea, loss of appetite, and irritability or apathy. There may be a moderate anemia, or this may be very decided, with great emaciation in advanced cases. Headache, vertigo and convulsions may occur, or paralytic symptoms or vague pains, especially abdominal. The temperature is normal or subnormal, or perhaps at times moderately elevated. One of the earliest symptoms in most instances is the pigmentation of the skin, varying from a very slight yellow to an almost-black color. Sometimes this is among the later manifestations. It is usually first seen in the face and hands, and is well marked about the genitals, umbilicus, arm-pits, nipples, and the flexor surfaces of the articulations. It may remain limited to these lesions, or be most marked here, but sometimes involves the whole surface of the body, even the hair. Upon the mucous membrane of the mouth and of the genitals there is a blotchy pigmentation. It is to be noted that some cases of Addison's disease exhibit little or no recognizable pigmentation of any region.

Course and Prognosis.—The duration is variable; sometimes 1 or 2 months, oftener a year or more. As the disease advances the asthenia increases, a cachectic state developing. There is rapid pulse and respiration, very low arterial tension, and subnormal temperature. Periods of improvement may sometimes occur, and rarely recovery has been reported; but there is some doubt about the latter, and, as a rule, the disease ends fatally by syncope, convulsions, or increasing cachexia.

Diagnosis.—This rests upon the combination of the pigmentation with the other symptoms described, and especially the very marked asthenia. Pigmentation of the skin occurring alone is not a certainly diagnostic symptom, since it may appear, for instance, in malaria, or from the continued administration of arsenic.

Treatment.—This is of little value. Efforts should be made to sustain the strength and to relieve gastrointestinal symptoms. Exercise must be avoided if there is much asthenia. Such tonic remedies as arsenic, phosphorus, and cod-liver oil may be employed. The administration of suprarenal gland may be tried; either fresh, or in the form of one of the extracts on the market. The results, however, have not been encouraging.

TUMORS OF THE SUPRARENAL GLANDS

Tumors of various sorts have been reported, among them sarcoma, carcinoma, adenoma, and mixed tumors. The pathological grouping of these growths is far from satisfactory. Provisionally the classification proposed by Glyn[1] may be adopted; viz. (1) *Benign tumors*, including hyperplasia, glioma and ganglion-neuroma occurring in the medullary portion; and diffuse hyperplasia, passing into adenoma, seen in the cortex. (2) *Malignant tumors*, of which there are two definite types; viz. sarcoma and allied growths, generally believed to be located in the medullary portion; and hypernephroma, situated in the cortex.

The symptoms produced by the malignant neoplasms vary considerably according to their nature and the portions of the organ involved, whether cortex or medulla, and for other reasons not well understood. Sarcoma and allied growths show a special tendency to metastasis, and do not produce the precocious sexual development which is so characteristic of cortical hypernephromata. The benign tumors are without characteristic symptoms.

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[1] Quarterly Journ. of Med., 1912, Jan., 157.

(*A*) **Sarcoma of the Suprarenal Gland.**—Growths of this nature are usually round-celled sarcoma or lymphosarcoma. They are uncommon but are seen oftenest in children. Frew[1] collected the reports of 51 cases, all in children and over half of them less than 3 years of age. The oldest case was 10 years; the youngest 2 weeks. More than half were in males. The primary lesion is a tumor which appears to arise in the medullary portion of the suprarenal gland, and to be of a sarcomatous nature; but of this latter there is doubt in some instances. Secondary growths resemble the primary in character. The distribution of these depends upon whether the left or the right gland is the primary seat. In the former there is involvement of the lumbar, mesenteric and post-mediastinal glands; those below the left sternoclavicular articulation; and some of those in the neck. The cranial bones are involved in nearly all cases, and sometimes the ribs. The long bones are not attacked, and the internal organs but seldom. An abdominal tumor may not be found until late in the disease. When the right suprarenal gland is the seat of the primary lesion, the growth is generally larger, and the discovery of an abdominal tumor is made early in the course. The process may extend to one or both kidneys. There is involvement of the lymphatic glands in the anterior mediastinum; at the right sternoclavicular articulation; above and within the liver; on the pericardium; above the upper surface of the right side of the diaphragm; and at the root of or within the lungs. The cranium is attacked in only about half of the cases.

Certain cases of sarcoma exhibit no very characteristic features, but in the others at least three distinct types can be recognized.

1. Malignant Suprarenal Sarcoma with Cranial Metastasis.—This is a remarkable clinical syndrome first described by Hutchinson.[2] Some of the cases of Frew's series belong here. Its characteristic feature is the involvement of the skull, which was present in all of Hutchinson's 10 recorded cases, 6 being his own; and in 4 others collected by Tileston and Wolbach[3] in addition to a new one reported by them. Tumors are also found in the adjacent lymphatic glands and in one or the other suprarenal body. The first *symptoms* are often misleading and are not referred to the abdomen, and the discovery of a tumor here may be made late or not at all during life. In some cases the first manifestations noted have been a discoloration of the eyelids, followed in a short time by exophthalmos, first on one and then often on both sides, the result of metastatic growths in the bones of the orbit. Tumors then develop elsewhere in the bones of the skull, especially in the temporal region. As the disease advances, the protuberance of the eyeballs becomes very great. Ulceration of the cornea may occur, followed by blindness; or this may depend upon optic neuritis. There is increasing anemia, exhaustion, and evidences of intracranial pressure. The *diagnosis* of the malady is rarely possible before the exophthalmos develops. When this has appeared, the possibility of scurvy with orbital hemorrhage is to be entertained; but this affection can be excluded by the lack of any of its other symptoms. Chloroma is eliminated by the absence of the characteristic alteration of the blood seen in that affection. No treatment is possible.

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[1] Quart. Journ. of Med., 1911, IV, 123.

[2] Quart. Journ. of Med., 1907–8, I, 33.

[3] Am. Journ. Med. Sc., 1908, CXXXV, 871.

2. Congenital Sarcoma of the Suprarenal Gland and Liver.—This is a type studied especially by Pepper[1] who collected 5 cases from medical literature, to which he added 1 of his own. Some of Frew's series of right-sided suprarenal tumors belong here. The lesions consist in the development of sarcomatous tissue arising either in the liver or in the suprarenal body. The malignancy is great, and the growth extends very rapidly, the liver becoming extensively infiltrated. In the suprarenal gland the growth is very hemorrhagic. There is great distention of the abdomen but no jaundice, pigmentation of the skin, or ascites, and but little pain. The course is short, death occurring within a few weeks.

3. Sarcoma of the Suprarenal Gland, with Metastases to the Long Bones.—This condition as described by Scudder[2] might be regarded as a third type. The cases reported by him occurred, however, only in adult subjects. There was a special tendency to metastasis in the long bones, although in a few instances other bones, as well as other tissues of the body, were also involved.

(*B*) **Cortical Hypernephroma.**—This unusual condition has been studied by a number of writers. Jump, Beates and Babcock[3] added an 18th to the 17 collected cases of Glyn.[4] All were confirmed by autopsy. Of these cases 1 came under my own observation; 15 were in females and 3 in males. The structure of this form of tumor is not uniform, and it is very probable that more than one variety of neoplasm is included under the title. In general the growth is characterized by the presence of large polyhedral epithelial-like cells of the nature of those found normally in the suprarenal cortex. Sometimes giant cells are present. The tumor grows much more slowly than is the case with sarcoma, and shows much less tendency to metastasis. The symptoms would appear to depend upon a disturbance of the function of the gland, probably of the nature of an increase of the internal secretion. They are of a very striking character, consisting chiefly in a precocity of the sexual characteristics, such as the appearance of hair on the pubis, in the axillæ, and sometimes on the face; occasionally a hyperplasia of the genital organs; and as a rule the production of obesity and sometimes of unusual general physical growth. It is noteworthy that there is a development of the male sexual characters only. Thus females show a tendency to the exhibition of the male characteristics, as seen in the unusual size of the clitoris and an absence, except in one instance reported, of early increase in size of the breasts. Males, however, do not show any of the feminine sex-characters, but rather a general early somatic precocity. There is no characteristic mental change. The cases have been divided by Guthrie and Emery[5] into two types:

1. The Precociously Obese Type.—This may affect either males or females. There is not true precocity of development of the generative organs, although the growth of the body-hair on the pubis and elsewhere is well-advanced. The obesity is a marked feature. The deposit of fat suggests that seen in adult life, the increase being especially prominent in the abdomen, chest, cheeks and neck; while the extremities remain thin.

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[1] Am. Journ. Med. Sc., 1901, CXXI, 287.
[2] Publicat. Mass. Gen. Hosp., 1907, I, No. 3, 82.
[3] Am. Journ. Med. Sci., 1914, CXLVII, 568.
[4] Quart. Journ. of Med., 1912, Jan., 157.
[5] Clin. Soc. Transac., London, 1907, XL, 175.

The features are dusky and congested, and often exhibit stellate veinules. There may be pigmentation, but no bronzing of the skin. The mammæ are large from the deposit of fat and the abdomen is prominent and pendulous (Fig. 417). The obesity is distinguished from that of hypopituitarism not only by its distribution, in that it is not especially in the breasts, mons veneris and hips as in the latter disease, but also by the absence of the sexual infantilism of the pituitary disorder. The obesity of pineal disease is less marked, and in that disorder there are also the evidences of intracranial tumor present.

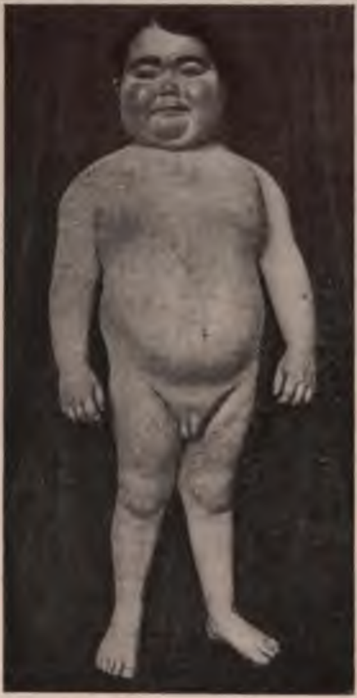

FIG. 417.—CORTICAL HYPERNEPHROMA WITH PRECOCIOUS OBESITY.

Boy, aged 4¾ years. Stoutness with growth of hair on face and pubis began 2 years before, cheeks large and bright red, whole upper part of the body fat, buttocks and legs not especially so, but muscular, genital organs of size for age. Died of acute tuberculosis. Autopsy showed hypernephroma. (*Guthrie and Emery, Trans. Clin. Soc. of London*, 1907, *XL*, 182.)

2. The Infant Hercules Type.—This is seen in males only. Here there is unusual development of the whole muscular and skeletal system, with actual sexual precocity. Obesity is absent. The case of Jump, Beates and Babcock[1] seems to occupy an intermediate position. Although a female, there was remarkable over-development of stature and muscular power without obesity and without any evidence of sexual precocity.

The *prognosis* of cases of cortical hypernephroma is unfavorable. All of those on record have died before the age of 16 years. The *diagnosis* is to be made chiefly from other conditions which produce early hirsuites. The chief diagnostic feature is the presence of the precocious hirsuites without true early sexual development, with the exception of some of the males; combined with the discovery of an abdominal tumor. Disease of the pineal gland, ovaries or testis may produce sexual precocity with hirsuites, while that of the pituitary body, although sometimes causing overgrowth of the body, leaves the generative organs and the sexual characteristics in an undeveloped state. *Treatment* is unavailing unless by early operative interference; which has not, however, as yet proven of benefit.

In this connection reference may be made to the fact that remains of the suprarenal tissue may often be found in other regions than the suprarenal body; as in the kidneys, ovaries, testes, broad ligaments, spermatic veins and elsewhere; and that a tumor of the nature of hypernephroma may develop with these remains as its base. Most frequent of these is the renal hypernephroma, which is of more common occurrence than the suprarenal growth, at least after childhood is passed. This tumor does not produce any alteration in the sexual characters.

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[1] *Loc. cit.*

HEMORRHAGE INTO THE SUPRARENAL GLAND

Etiology.—This comparatively uncommon affection is rendered readily possible by the vascularity of the suprarenal body. Hamill[1] reported on 90 cases including 3 of his own, occurring in the new born, and autopsies systematically made on new-born and especially still-born children would doubtless greatly increase the number of cases. At this period the hemorrhage may be the result of the influence of long-continued or difficult labor, or of septic infection through the umbilicus. In older subjects it may attend the acute infectious or other febrile disorders, accompany purpura hæmorrhagica, or be due to some entirely obscure cause. It is rarely seen beyond the 1st year of life.

Pathological Anatomy.—The hemorrhage may be bilateral or unilateral, in the latter event being oftenest upon the right side. The affected gland is enlarged and frequently greatly so, dark-red in color, or in cases where the hemorrhage is of quite moderate amount the capsule still preserving its yellow tint. Section shows that the hemorrhage is situated especially in the medullary portion, the glandular tissue being more or less destroyed; and in severe cases the entire gland is transformed into a sac containing blood. Frequently blood has escaped into the peritoneal cavity. Thrombosis of the renal vein or of the inferior cava has been noted.

Symptoms.—These are always obscure, and as a rule the diagnosis can be made only at autopsy. In the new born there is sudden onset, with abdominal pain, fever, and collapse; the duration of the case varying, but lasting not over a few days with symptoms of peritonitis. Oftener death seems entirely sudden, probably from severe intraperitoneal hemorrhage. When occurring in older infants there is the sudden onset of an acute febrile process, perhaps accompanied by vomiting and diarrhea. A purpuric eruption may occur, and collapse terminate the process after 24 to 48 hours. Cases of this type sometimes occur almost epidemically. The prognosis is hopeless, and treatment unavailing.

OTHER LESIONS OF THE SUPRARENAL GLANDS

Similar to the changes produced by cortical hypernephroma are those resulting from a benign **hyperplasia** of the suprarenal cortex. In pseudohermaphroditism of the female type, with development of the organs suggesting the male characteristics, there may be associated a congenital hyperplasia of the suprarenal cortex. On the other hand, a **hypoplasia, atrophy,** or **absence** of the suprarenal glands may be productive of symptoms exactly the reverse of those seen in cortical hypernephroma. The condition is liable to be associated with failure in the development of the brain, especially anencephalus. **Syphilis** may produce interstitial changes in the suprarenal glands, and **tuberculosis,** as stated, is the most frequent cause of Addison's disease.

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[1] Arch. of Ped., 1901, XVIII, 81.

CHAPTER IV

DISORDERS ATTRIBUTED TO THE INTERNAL SECRETIONS

There are certain disturbances of growth or nutrition which appear to be clearly the result of a diseased state of one or more of the internal glands; and others where an interference with any glandular function can be only assumed to be probably present. Certain cases are encountered which seem to be dependent upon such disorders, while others with a very similar clinical picture are clearly not so related. The conditions produced by disorders of secretion of the pineal gland, the suprarenal bodies, and the thyroid gland have already been discussed (pp. 523, 524, 509, 515). To be considered as dependent, at least in many instances, on a disturbance of function of some of the internal glands are *Infantilism; Obesity; Lypodystrophy* and *Precocity*.

INFANTILISM

That all forms of infantilism are dependent upon disorders of the internal secretion is more than doubtful. That a number of them, however, are produced in this way is beyond question and all may be studied together as a matter of convenience.

The term "Infantilism" is employed to designate a persistence to a greater or less extent of the bodily and often of the psychic characteristics of infancy or childhood. In a strict sense this can apply only to those who have attained adolescence or adult life; but it is equally true even of infancy, the condition being then a relative one; viz. a persistence of characters belonging to a time of life decidedly earlier than the actual age of the patient. Further, it is not so much the arrest of growth as the retention of some of the somatic characteristics of early life which should constitute infantilism. Thus there may be dwarfs (*nanism*) who are in this respect not infantile, as, for instance, subjects of severe rickets, achondroplasia, etc. There has been an arrest of *growth* but not of *development* (Gilford).[1] More rarely there may be cases of infantilism which are not dwarfs, as in some instances of pituitary infantilism, the subjects exhibiting the infantile development of the genital organs, or being infantile only in certain other respects. Yet there is much confusion in the classifications which have been employed; and, omitting certain of the types of nanism, and with the reservation that infantilism and nanism are in reality different, a discussion of the topic can be held under the general heading of Infantilism.

Confusion, too, exists in the differentiation of the forms of infantilism from each other. The subject has been studied with care by Hastings Gilford[2] and a modification of the classification as suggested by him may serve the present purpose. This divides the disease into cases of (*A*) *The Essential group,* including ateleiosis and progeria; the causes being entirely unknown, and the condition being apparently a freak of development; and (*B*) *The Symptomatic group,* which includes all the remainder; the cause here being some disturbance of function or other factor to which the infantilism is secondary.

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[1] Lancet, 1914, I, 587.

[2] *Loc. cit.*

(*A*) Essential Infantilism. **1. Ateleiosis.**—A prominent characteristic of this condition is the inherited and familial predisposition. No direct cause is discoverable. There is an arrest of development occurring generally in infancy or early childhood, and the subjects are consequently dwarfed. The infantile characteristics which existed at the time of onset persist to a large extent. These are shown in the short limbs, the large head, the small face, the shape of the abdomen and chest, and the general roundness of form. These are the most typical cases of infantilism. Gilford divides them into the *asexual* and the *sexual* types, although the distinction cannot always be sharply defined. In *asexual ateleiosis* there is a very marked delay in the development of the whole body, shown in some parts more than others, the sexual organs being the most backward of all. When the time of puberty arrives, no change in the sexual characters takes place, much of the epiphyseal and other bony alterations do not occur, and growth goes very slowly onward, continuing well into adult life, the other general somatic characteristics of childhood persisting. In some instances the sexual organs eventually do mature and such cases can then no longer be called asexual infantilism. There is no involvement of the intellectual power. In *sexual ateleiosis* the infantile features continue until the time of puberty comes, which, however, is often much delayed. Then the sexual organs and powers develop normally, the osseous changes natural to this process take place, and further growth of the body ceases; the patient becoming a miniature man or woman; yet in physiognomy, proportions, and size still a child. Many of the professional dwarfs belong to one or the other of these classes; as, for instance, General and Mrs. Tom Thumb and others.

2. Progeria.—This is a term designating premature old age. It is a very uncommon condition, of which, however, some remarkable instances have been reported (Gilford). In it there is a combination of very decided infantilism with premature decay, as shown by the early development of white hair or of baldness; arteriosclerosis; emaciation; wrinkling of the skin; and the general carriage and appearance of an old man or woman, although in actual age the patient is no more than an adolescent or young adult.

(*B*) Symptomatic Infantilism.—This differs from the essential type described not only in the less conspicuous arrest of development and persistence of child-like somatic characters, but in the absence of any familial predisposition and in the fact that the cases are always secondary to some previous morbid condition. Various types have been described, many of them conflicting. Basing the division on the clinical appearances, two principal forms are ordinarily recognized:

1. The Lorain Type.[1]—In this the subject is small and looks at first sight like a child; but examination shows that the proportions and outlines are more truly those of a miniature adult. The head is small; the limbs slender, but normally long in comparison with the trunk; the chest and abdomen have the shape and proportions of an adult. There is, indeed, nothing of the child-form except the shortness in height and the entire failure of development of the genital organs and of other sexual characteristics commensurate with the age; although properly porportioned to the size (Fig. 418). The intellect may be normal or its development retarded. There is often debility and the general health is poor. These subjects, then, in their final stage represent in some respects a condition

[1] Lorain. Preface de la Thèse de Faneau de la Cour., Paris, 1871. Ref., Apert in Traité des mal. de l'enf. (Grancher, etc.), 1904, I, 993.

of nanism rather than infantilism; the characteristic features of infancy or childhood having disappeared, except the lack of sexual development and the size of the body. Among etiological factors of importance are hereditary syphilis; the lymphatic diathesis; any cause tending to produce chronic malnutrition, such as malaria, tuberculosis, defective hygiene, insufficient or improper food; congenital or acquired disease of the heart; arterial hypoplasia; or chronic poisoning by substances which have been

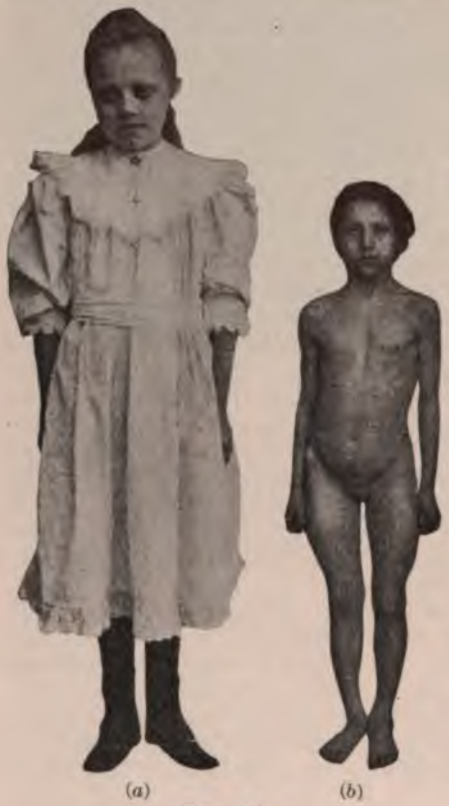
(*a*) (*b*)

Fig. 418.

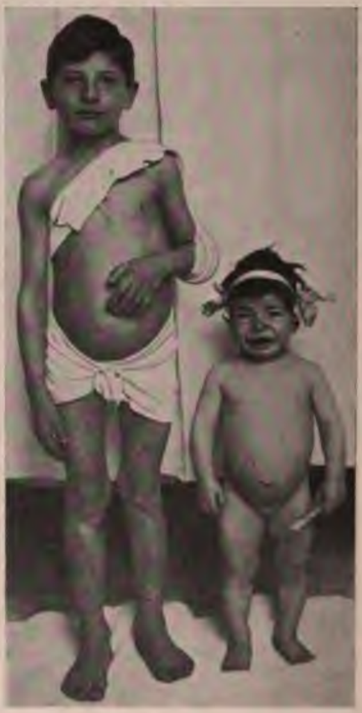
Fig. 419.

Fig. 418.—The Lorain Type of Infantilism.

Child of 12 years (*b*) is standing by a normal girl of 10 years (*a*). (*Peritz, Ergebn. der inn. Med. u. Kinderh.*, 1911, *VII*, 457.)

Fig. 419.—Infantilism, Brissaud's Type.

Marie G., admitted to University Hospital, Philadelphia, Jan. 23, aged 7 years. Child normal to 21 months when had very dangerous attack of pertussis. Since then has grown very little. Said to be normal mentally. Examinations showed extremities short for size of thorax, legs bowed, skin harsh and dry, head large, hair of scalp short and moderately harsh, eyes somewhat far apart, palpebral fissures narrowed, tongue looks slightly larger than normal, but does not protrude, thyroid gland cannot be felt, and thyroid region seems less full than normal. Slight subcutaneous deposits of fat in supraspinous fossæ, abdomen greatly distended. Temperature averages below normal. Height 29 inches (74 cm.), weight 20 pounds (9072 gm.). Feces negative for bacillus bifidus, bacillus infantilis, coccal forms and fermentative forms. *X*-ray examination negative. Condition practically uninfluenced by thyroid extract, thymus extract and pituitary extract. Photograph shows comparison with boy of same age and of normal development, with a height of 48 inches (122 cm.). (*Griffith, Amer. Jour. Dis. Child.*, 1918, *XVI*, 103.)

introduced from without, such as tobacco, opium, and lead, or produced within, as in chronic autointoxication. In many cases no cause whatever can be discovered.

2. The Brissaud Type.[1]—In this form the body is small, the head large, the abdomen prominent, the limbs short and rounded, the trunk relatively large, there is a tendency to obesity, the sexual organs are undeveloped and the somatic proportions are infantile (Fig. 419). The intellectual development is retarded. This condition has sometimes been named *myxedematous infantilism* and has been attributed to hypothyroidism. This is doubtless the cause in many cases; while in others the condition is considered to be dependent upon a disturbance of the pituitary secretion. It has been found that an influence upon development may occur as a result of hypothyroidism without there being any marked myxedematous symptoms discoverable (p. 512); and the administration of thyroid substance has sometimes benefited infantilism, not only of the Brissaud but of the Lorain type.

These two clinical types, and especially that of Lorain, cover to a large extent many of the other forms of symptomatic infantilism, which may be named more correctly on an etiological basis. Classified in this manner the following may be mentioned:

3. Intestinal Infantilism.—In infantilism of this variety, studied particularly by Herter,[2] there is arrest of growth, with emaciation, anemia, fatigue on slight exertion, abdominal distention, at times diarrhea, and the presence often of a large amount of fat in the stools, especially in the form of fatty acids or soaps. The feces are whitish in color, gruel-like in consistence, and of an offensive odor. The appetite is variable or often excessive, and thirst decided. The urine is increased in amount and shows among other abnormalities an excess of ethereal sulphates. The intellect is normal, or the child even precocious. The disease begins oftenest in the 2d or 3d year of life, and may last several years, with periods of improvement followed by relapse. Herter ascribed the condition to an absence of the normal intestinal flora and the presence of Gram-positive organisms in great excess. The bacillus bifidus, bacillus infantilis, and coccal forms characteristic of infancy are still present, while the bacillus coli and bacillus lactis aërogenes are absent.

The condition is, in fact, a chronic intestinal infection which results in interference with nutrition and a starving of the tissues; the disease being an "infantilism by deprivation," according to Gilford's classification; perhaps increased by the toxins produced.

4. Pancreatic Infantilism.—This form of infantilism was so named by Bramwell.[3] It is certainly closely allied to Herter's type, and the general symptoms are very similar. The diagnosis in the case reported by Bramwell rested upon the remarkable improvement which followed the administration of pancreatic extract. This patient 18 years of age at the time of first observation had a development of the generative apparatus similar to that of a boy of 11 years.

5. Pituitary Infantilism.—The disorder receiving this title was first described by Fröhlich,[4] and often called after him "Fröhlich's Syndrone;" or "dystrophia adiposo-genitalis" (Bartels).[5] It depends upon the presence of a tumor or some disturbance of function of the pituitary body resulting in hypopituitarism. In a case reported by Madelung[1] there was a gun-shot wound of the sella turcica. The symptoms, apart from those indicating a lesion of the hypophysis, such as headache, bitemporal hemianopsia and exophthalmos, are those connected with development. The sexual organs remain infantile in character; or, in the case of patients where the disease began in adult life, undergo a reversion to some extent to the infantile condition. There is no hair on the pubis or in the axillæ. A characteristic symptom is the marked tendency to obesity, which is general, although most pronounced on the trunk, mammæ, mons veneris and hips. Polyuria has repeatedly been observed. There is an unusual tolerance for carbohydrates, and very large amounts of sugar can be ingested without producing glycosuria. The temperature is often subnormal. There is general sluggishness, drowsiness and asthenia. Retarded skeletal development is a feature, but it is possible for the reverse of this to occur, as observed in a case of my own (Figs. 420 and 421). This may depend, as Cushing[2] suggests, upon activation of the anterior lobe combined with insufficiency of the posterior lobe.

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[1] Brissaud, Leçons sur les mal. nerv., 1895, 625.

[2] Trans. Assoc. Amer. Phys., 1910, XXV, 528. Intestinal Infantilism, 1908.

[3] Scottish Med. Journ., 1904, XIV, 321.

[4] Wien. klin. Rundschau, 1901, XV, 883.

[5] Zeitschr. f. Augenheilk., 1906, XVI, 407; 530.

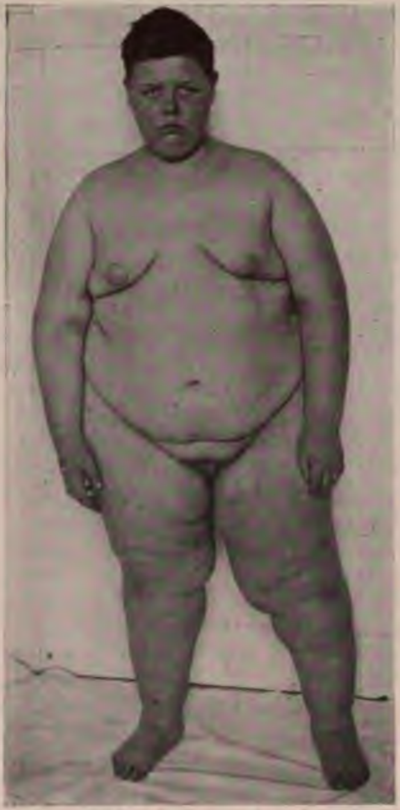

FIG. 420.—PITUITARY INFANTILISM.

John S., aged 11 years. Admitted to University Hospital, Feb. 7. The tendency to obesity appeared at the age of 11 months, and has increased since then. Is mentally bright. Height 5 feet, 1 inch (160 cm.), weight 251 pounds (113,851). Penis small for his age and very small in comparison with his other development, buried in fat, testicles descended and very small, almost no trace of pubic hair. *X*-ray examination by Dr. Pancoast showed sella turcica definitely smaller than normal, indicating a small pituitary body. (See Fig. 421.) Sugar tolerance increased, but this diminished markedly after continued administration of pituitary extract. Prolonged treatment no practical effect on the obesity. (*Griffith, Amer. Jour. Dis. Child.*, 1918, *XVI*, 103.)

Among other forms of infantilism may be mentioned *hepatic infantilism*, in which the general condition of infantilism with stunted growth is associated with cirrhosis of the liver. A number of such cases have been collected by Lerebouillet.[3] Another variety is *renal infantilism*, dependent upon a chronic interstitial nephritis, polyuria being a symptom in such cases, as it is, indeed, in some of the instances of infantilism due to other causes (Fletcher,[4] Parsons,[5] Langley Porter[6]). A *cardiac infantilism* has also been described, dependent upon hypoplasia of the heart and arteries, as in some of the cases of Lorain's type, as described by Parkes Weber[1] and others. There is also an infantilism apparently connected with the *status lymphaticus*, in which sometimes hypoplasia of the genital organs and obesity persist after the age of puberty. That diseases of the *thyroid gland* produce stunting of growth and failure of development is well known, as in cretinism, and as in Brissaud's type of infantilism and probably in other forms; and it is very likely that under certain conditions affections of the *thymus gland* or of the *suprarenal bodies* may be active etiological factors. Brilliant results have followed in a few cases of infantilism the administration of extract of the suprarenal gland. The influence of the early removal of the *testicles* upon development of sexual characteristics is well known, and some cases of infantilism, at least in certain somatic characteristics, may depend upon a disturbance of the internal secretion of these organs by some pathological process; although in length of body gigantism is often produced. In most instances of infantilism the interdependence of the various internal glands is probably a factor in the production of infantile characteristics.

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[1] Arch. f. klin. Chir., 1904, LXXIII, 1066.

[2] The Pituitary Body, 1912, 177.

[3] Les cirrhoses biliaires, 1902, 76.

[4] Proc. Royal Soc. of Med., 1911, Sect. of Dis. of Child., 95.

[5] Brit. Med. Journ., 1911, II, 481.

[6] Arch. of Pediat., 1915, XXXII, 85.

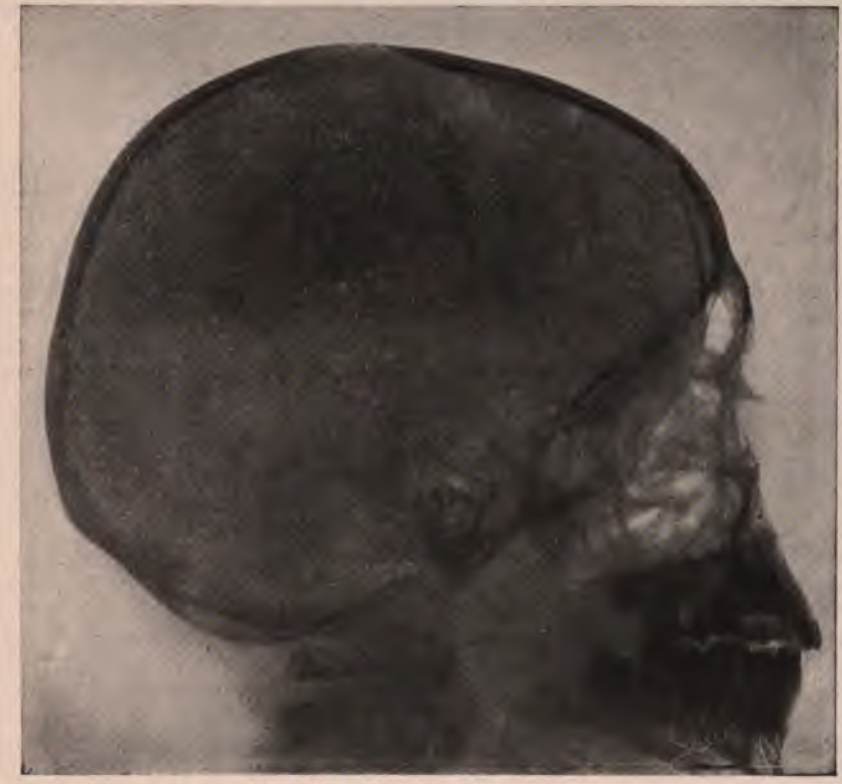

FIG. 421.—PITUITARY INFANTILISM.
Radiograph of the head showing small cella turcica. Same case as Fig. 420.

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[1] Brit. Jour. Child. Dis., 1913, X, 203.

In addition to these varieties of infantilism there are others, more strictly speaking forms of **nanism**, to which the title *Simple Correlative Infantilism* has been applied. Here, together with some other forms, Gilford places cases of the failure of bodily development, at least in some respects, seen in achondroplasia, muscular dystrophy, hydrocephalus, microcephalus, and mongolism.

The **treatment** of infantilism is that directed to the cause. Thus Bramwell's case of pancreatic infantilism was aided by the administration of pancreatic extract; cases of the Brissaud type are amenable to thyroid extract; the administration of pituitary extract has benefited subjects with the Fröhlich's syndrome; and the correction of the intestinal condition is indicated in Herter's type.

OBESITY

(Adipositas)

It would seem certain that not all cases of obesity are dependent upon disorders of the internal secretions; but inasmuch as it is equally beyond dispute that others of them are, they may well be considered together in this connection. As a symptom the condition has already been referred to in discussing certain of the endocrine glands. Many infants and children have a tendency to develop abundant adipose tissue which is to be considered entirely within physiological limits. The boundaries between this and a pathological obesity are naturally not sharply defined. The limit may be placed at a point where the fatness begins to be a deformity or a cause of inconvenience. Many remarkable cases are on record occurring even in infancy, although oftener in later childhood. One of the important studies of this subject is by Braoude.[1] Among those collected by him are some striking instances of congenital obesity, as, for example, that reported by Chambers of an infant weighing 16 pounds (7257) at birth; and another by Clairin[2] weighing 13 pounds (5897). Some of the cases should probably be regarded precociously large rather than obese. (See Precocity, p. 538.) Heubner[3] observed an infant weighing at the 8th month of life 18.5 kg. (40.78 lb.). After the period of infancy has been passed we find such cases as that reported by Marcé of a child of 13 years weighing 214 pounds (97069); Hilbairet, one of 5½ years of 127 pounds (57606), and Percy and Laurent of one of 4 years of 150 pounds (68039). Marked obesity at this time of life is, however, not common. A few instances of hemi-obesity in early life have been reported, as in the cases of Shaw[4] and of Hutchinson;[5] the autopsy in the last showing a probable dependence upon asymmetrical development of certain of the internal glands.

Etiology.—Various causes may be operative. Heredity is an uncommon one in children, although its influence is frequently seen in adult life. The commonest factor is the diet. In one case of Heubner's,[6] an infant weighing 10.44 kg. (23 lbs.) at 5 months, there was a hearty appetite, the maternal secretion was unusually rich in fat, and the estimated calories of the milk taken per day equalled 900, an amount much in excess of the normal requirements. In the obesity of later childhood, seen rather oftener in females, diet plays a prominent part also, the food containing a large excess of fat or of carbohydrate, or being in all particulars far too abundant. With the factor of diet is naturally to be studied the amount of exercise taken. A child who is very active will tolerate an amount of food which in a less energetic subject might produce obesity. Apart from this it seems possible that there may be operative a lack of balance in the metabolic processes, entirely independent of the amount of exercise taken, and not associated with the ingestion of an undue amount of food. There will thus be certain children who show a special tendency to become obese without discoverable cause. This question has not yet been settled. The results of the experiments of Rubner[1] are opposed to this view. In any event it is certain that once obesity is established it is liable to persist unless means are taken to relieve it. Not only does the condition make the patient incapable of taking sufficient exercise, but the ratio of the surface of the body to the body-weight is less than in the normal child, and a proportionately smaller amount of the energy of the food is dissipated here.

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[1] Thèse de Paris, 1900.
[2] Thèse de Paris, 1855.
[3] Kinderheilk., 1911, II, 52.
[4] Proc. Roy. Soc. Med., 1914, VIII, 1, 15.
[5] Brit. Journ. Child. Dis., 1904, I, 258.
[6] *Loc. cit.*

In addition to the causes mentioned we find early obesity an attendant upon certain definite morbid conditions. It is sometimes seen in subjects of lymphatism, and is a very prominent symptom of cretinism, although the apparent fatness is to a considerable extent dependent upon the myxedematous condition of the skin. It is also one of the characteristics of the disorder of the pituitary body described under infantilism as dystrophia adiposo-genitalis. Here it is associated with retardation in the development of the genital organs and functions. In combination with premature sexual development it may be seen in hypernephroma; and it has been reported also in some instances of tumor of the pineal gland. The influence of the testis in producing obesity is observed in some cases of castrated individuals.

Symptoms.—The deposition of fat follows other lines than those seen in middle age. There is not the special tendency to abdominal fat, but a more uniform distribution. The face is full and round; the cheeks and eyelids full; the upper and lower part of the trunk affected alike; the mammæ in both sexes large from the deposition of fat; the extremities fat. This description does not apply so well, however, to some of the cases of obesity dependent upon disordered internal secretion. Thus in Frölich's syndrome (p. 533) the fat is found chiefly on the thorax and abdomen, and especially in the breasts and the mons veneris, although the rest of the body is also obese; while in that associated with hypernephroma the extremities may be thin, and the adiposity is seen chiefly in the trunk, neck, and in the bloated congested cheeks and pendulous prominent abdomen.

Treatment.—Apart from the cases due to diseases of the internal glands, obesity in children lends itself readily to treatment. This consists chiefly in a modification of the diet, of such a nature that the tendency to accumulation of fat may be checked; yet not of such a character that the normal strength and development of the patient shall be influenced. In some instances of obese breast-fed infants the excessive deposit of adipose will disappear of itself when weaning takes place. This was true, for example, in one of Heubner's cases. In general the amount of carbohydrate in the diet, and especially of the fat, should be decidedly reduced, and the total amount of nourishment as well; but this should be done gradually and cautiously, watching carefully, meanwhile, the alterations in weight which follow. A diet of green vegetables and fruit should replace to a considerable extent that of carbohydrates; yet these latter cannot be reduced too greatly, lest a failure to assimilate protein in sufficient quantity occurs and the health suffer in consequence. The diet cannot be made as rigid as in adult life. A diminution in the quantity of liquid ingested may also be of service. A decided increase in the amount of exercise taken is to be insisted upon. This is sometimes difficult to manage, and it may require considerable thought to devise the best method. Thyroid extract should be administered only in cases where hypothyroidism is suspected. In the obesity of hypopituitarism extracts of the pituitary body have been given with advantage, and sometimes those of the thyroid and adrenals are of service.

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[1] Ernährung im Knabensalter, mit besonderer Berucksichtigung der Fettsucht, Berlin, 1903.

LIPODYSTROPHIA PROGRESSIVA

This little known and uncommon affection was described under this title by Simons,[1] although a similar case had been earlier reported by Campbell.[2] Feer[3] gives illustrations of 2 cases in children, and Herman[4] published the first case reported in the United States. A very thorough review is made by Parkes Weber.[5]

Etiology.—The cause is entirely obscure, but is probably some disorder of the endocrine glands. All of the earlier cases were in females, and the affection was supposed to be confined to that sex; but undoubted examples in males have been observed. Boissonnias[6] records an instance in a boy of 6 years. The large majority of the cases have been observed in adults, but their histories show that the affection usually began in childhood, generally about puberty.

Symptoms.—These consist in a progressive and finally complete wasting of the subcutaneous fat of the face, arms and trunk. The tissue of the skin is entirely normal. The face is the part usually first affected, and the advance to other regions is generally slow. A characteristic of the affection is the over-development of the fat of the buttocks, hips and thighs, and to a less extent of the calves. This generally does not begin until several years after the onset of the disease. There are no other symptoms, and the general health and strength is in no way affected, nor does the duration of life appear to be curtailed. The diagnosis offers no difficulty, at least after the increase of fat of the lower part of the body has begun. Treatment has been of no avail.

PRECOCITY

Mental precocity has already been discussed under Diseases of the Nervous System (p. 297). In this condition is considered only precocious development of the body or its functions, in whole or in part. This is oftener seen in females, and is then represented either by precocious menstruation, which may begin in the early months of life and continue with more or less regularity; or by a more general precocious maturity. In the former early menstruation is the only characteristic, the subjects remaining infantile or child-like in most other respects to a degree corresponding to the age. In the latter there may be in some cases precocious sexual maturity only in the condition of the genital organs in addition to menstruation; in others the children may be over weight at birth, there is a rapid growth of the whole body in height and weight, with premature development of the sexual organs and breasts and general sexual characteristics. As regards the excessive birth-weight, some of the cases referred to under Obesity (p. 536) probably belong to the category of early precocious development of the whole body. The most remarkable instance published is that reported by Belcher[1] of an infant which not only weighed 25 pounds (11349) at birth but measured 28 inches (71 cm.) in length. Menstruation in subjects of precocity is early, but is antedated by some of the other evidences of premature development. The intelligence, too, may be above normal for the age (Morse), but is not always so. The sexual desires are often early developed. In more than one instance on record pregnancy has occurred at the age of 8 years. In male subjects there may be emissions of semen as early as 2 or 3 years of age. The subject has been especially studied by Gautier,[2] Morse,[3] and Lenz.[4] The last mentioned writer has collected 130 cases of precocious menstruation, most of them showing other signs of precocity as well. (See also Genital Hemorrhage, p. 230.)

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[1] Zeit. f. d. gesammte Neurol. u. Psych., Orig., 1911, V, 29.
[2] Trans. Clin. Soc., London, 1907, XL, 272.
[3] Jahrb. f. Kinderh., 1915, LXXXII, 1.
[4] Arch. Int. Med., 1916, XVII, 516.
[5] Quart. Journ. of Med., 1917, X, 131. Brit. Journ. Child. Dis., 1917, XIV, 81.
[6] Ref. Pediatrics, 1917, XXIX, 94.

The typical cases of premature menstruation or of precocious maturity have no discoverable cause. There are, however, others in both sexes in which a condition of premature sexual development is dependent upon some disease of the internal secretions. Thus it is seen in cases of hypernephroma, and in tumors of the pineal gland or of the ovary. In males suffering from hypernephroma there may be in addition to the early sexual development a remarkable growth in stature and of muscular power without obesity, producing what has been called the Infant Hercules type. (See p. 528.) In some cases in females there may also be an unusual development of the body in general, with the early appearance of pubic hair. As a rule, however, these female patients do not show any evidence of actual precocious sexual conditions. Gigantism can be called a form of precocious development of the body so far as growth in height is concerned. In other respects some of these subjects of the eunuchoid type are the reverse of precocious, being sexually undeveloped.

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[1] Journ. Amer. Med. Assoc., 1916, LXVII, 950.
[2] Rev. méd. de la Suisse Romande, 1884, IV, 501.
[3] Arch. of Ped., 1897, XIV, 241.
[4] Arch. f. Gynäkol., 1913, XCIX, 67.

SECTION XIX

DISEASES OF THE SKIN, EYE AND EAR

CHAPTER I

DISEASES OF THE SKIN

Dermatology as it applies to early life is of itself a subject so large and requiring such special knowledge that no attempt can be made here to consider it at any length, in spite of the very great frequency of cutaneous diseases in infancy and childhood. Only those disorders will be discussed which are either entirely or largely limited to early life, or which are of very frequent occurrence at this period or exhibit special features incident to it. In all the description must be brief; and for fuller details works on dermatology must be consulted. The confusion attending the classification of the affections of the skin is so evident from the different systems employed by writers, that the divisions and sequence used in the following descriptions are largely arbitrary and a matter of convenience. I have made free use of various publications upon dermatology; and in this connection I would express my indebtedness especially to the text-books on dermatology of my past and present colleagues at the University of Pennsylvania, Drs. Duhring, Van Harlingen, Stelwagon, Hartzell, and Knowles.

SUBCUTANEOUS EMPHYSEMA

Brief attention must be directed to the fact that air may accumulate in the subcutaneous tissue as a result of various causes. Developing in the eyelids it may result from fracture of the orbit, permitting air to enter from the sinuses. In the neck it may follow tracheotomy, deep ulcerative processes in the mouth or pharynx, wounds of the esophagus, or ulcer of the stomach; or oftener be an extension from emphysema of the connective tissue of the mediastinum. Appearing primarily in the thorax it may be the result of a rupture of an emphysematous or tuberculous pulmonary lesion, combined with local obliteration of the pleural sac; and repeatedly it has been caused by exploratory puncture of the pleural cavity, under the same condition of obliteration, in which the needle has entered the pulmonary tissue. The direct cause in many instances is the occurrence of violent cough or severe dyspneic respiration. In the case of exploratory puncture, ulceration, or other trauma, the air passes at once into the adjacent subcutaneous tissue. In the more numerous cases of subcutaneous emphysema, especially when the condition is associated with pulmonary emphysema or tuberculosis, or is of unknown origin, the air reaches first the cellular tissue of the mediastinum and makes its way thence to the subcutaneous tissue of the neck, and perhaps quite extensively over the body. An entirely different class of cases is that dependent upon the production of gas subcutaneously in certain forms of septic infection. This condition is not considered here.

Symptoms.—These consist in a swelling of the skin, which on palpation is found to yield readily to slight pressure, with the development of a peculiar sensation of crepitation as felt by the finger. The swelling is generally first detected in the lower part of the neck. It may remain slight and limited to this region, or may spread with considerable rapidity in different directions, reaching upward and producing great swelling of the face and head, and extending downward to the thorax or below this. Sometimes it becomes almost universal. There appear to be no subjective symptoms which can be attributed to the condition itself. The duration is rather short, the air being absorbed gradually and a normal condition being attained after a week or slightly longer. Yet the **prognosis** for life is not good, inasmuch as this symptom is often an indication of some serious disease in connection with the lungs; often tuberculosis. Death may take place soon after subcutaneous emphysema develops, but is brought about by the primary disease. Treatment is not indicated.

EDEMA

Cutaneous edema is, of course, only a symptom produced by a large number of etiological factors. Its importance is such that a brief review of these will be of advantage.

Rarely there is a *fetal edema*, seen as a general dropsical state, perhaps associated with malformations or diseases of the organs, although none of these appear to be certain etiological factors. In some cases pathological conditions in the mother may be the cause, such as disease of the heart or kidneys, or some disorder of the placenta. Generally the infant is still-born, or lives at most a few days. *Edema neonatorum* is a term applied to the transitory cutaneous edema seen sometimes in the early weeks of life. This affection has been described under Diseases of the New Born (Vol. I, p. 301). Perhaps the most frequent cause of edema in early life is *nephritis* (see p. 176), a disorder not uncommon even in infancy; at which time also the influence of *gastrointestinal diseases* and of *syphilis* in the production of dropsy is not to be forgotten. Occasionally an edema having the distribution and appearance of that of nephritis develops without the presence of albumin or of formed elements in the urine; and it is uncertain whether the cause is an inflammation of the kidney or is of a toxic nature, often arising from the gastrointestinal canal. Such a condition has been reported after scarlet fever by a number of observers. With Dr. Wm. S. Newcomet[1] I have reviewed the subject some years ago. *Cardiac edema* is common, but less so than in adult life. *Marantic edema* is a frequent symptom in much debilitated infants; and in older children as well, when exhausted from any cause. It is most marked in the hands, feet and face, but may become widespread. The affected parts pit easily and are cold to the touch. Severe *anemia* is another fruitful cause of edema, and the condition may also attend *purpura*. There occurs also an "*essential edema*" in which ascites is combined with hydrothorax and general anasarca, without any discoverable cause.

Localized edema is seen in *erysipelas*, and may sometimes persist in cases occurring in early infancy after the original disease has disappeared. So, too, a localized edema of slight grade is seen in the face in *measles*, and about the eyelids in severe *pertussis;* and in various parts of the body as a result of *mechanical interference* with the circulation, as by tumors or enlarged glands. A circumscribed cutaneous swelling is a characteristic of *angioneurotic edema*. (See p. 544.) *Tetany* is frequently accompanied by edema of the dorsum of the hands and feet, perhaps through mechanical obstruction of the vessels by the spasm, although this is not certain. A *persistent localized edema*, either congenital or acquired, may occur, and in some instances is hereditary, as in the remarkable family reported by Milroy[1] in which 22 out of 97 individuals in 6 generations had had permanent edema of both legs, congenital in origin except in 1 instance. I have observed a somewhat similar case (Griffith and Newcomet)[2] in which edema of the left leg and face, not pitting on pressure, began at the age of 3 months, and had been persistently present when the patient was examined at the age of 4 years. Some reported instances of what appeared to be persistent edema of the legs were probably dependent upon *elephantiasis*. There has been 1 such case under my observation.

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[1] Medical News, 1892, Oct. 2.

URTICARIA

(Hives)

Etiology.—This very common cutaneous affection may make its appearance at any age, and depend upon a variety of causes. A hereditary disposition is seen in some instances. Much the largest number of cases in early life are connected with the diet. Attacks of indigestion may be attended by hives; but independently of this many children exhibit an idiosyncrasy toward certain articles of diet, and suffer from the eruption without any digestive symptoms being produced. These articles vary with the individual, but among those oftenest producing urticaria are to be mentioned shell-fish, strawberries or other fruit, cheese, pork, sausage, eggs, and food not entirely fresh. In some of these cases the eruption appears to be an anaphylactic reaction to certain proteid substances. Intestinal worms are occasional causes, and a rather long list of medicaments may in some individuals evoke the disorder. (See Drug Eruptions, p. 549.) The hypodermic injection of a foreign blood-serum may produce a severe attack (see Diphtheria, Vol. I, p. 464), without doubt here being due to the protein. Urticaria may also result from cutaneous irritation of the body by fleas, certain hairy caterpillars, jelly-fish, nettles, and the like; and in those predisposed may even be caused by stiffly starched or woolen garments.

Pathology.—The typical wheal consists of a circumscribed edema chiefly in the upper layers of the skin, with dilatation of the blood-vessels and a certain degree of leucocytic infiltration. The disturbance is one of the vasomotor system, probably oftenest brought about through the action of some toxic substance.

Symptoms.—In typical cases these consist in the production of wheals. These are pale-red or white, round or oval, flattened elevations varying much in size. They may appear with the greatest suddenness, and last but a few hours, or a shorter or slightly longer time. The lesions are few or extensively distributed, and may come in crops in different portions of the body. Severe itching and burning attend the eruption and symptoms of associated indigestion may be present. The duration of the attack may be several days, fresh wheals being produced by the irritation from scratching; or through the development of repeated crops the disorder may assume a chronic condition.

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[1] Omaha Clinic, 1892, V, 101.

[2] *Loc. cit.*

This description of a typical case must be modified in many instances, and this is especially true of young children, and most of all of infants. Here there is a clearly emphasized tendency for papules, papulovesicles, vesicles and sometimes pustules to appear with or upon the wheals; and to this combination the titles *urticaria papulosa; lichen urticatus, red gum; strophulus,* etc., have been applied. Inasmuch as the wheals in this form may disappear early, or be present solely at night leaving only the other lesions discoverable by day, the diagnosis may be obscured. This variety of the disease is frequent and itching is very troublesome. The eruption may be widespread, but is especially liable to appear on the extensor surface of the arms and thighs, the lumbar region, buttocks, and the back of the hands and wrists. The lesions may be few and scattered or very numerous. Crusting and scabbing may result from repeated scratching. The title *urticaria pigmentosa* is applied to a rare condition in which urticarial wheals persist as pigmented areas.

As **complications** of urticaria may be mentioned exudative erythema, purpura, and arthritis; one or all of these being attendant. (See Purpura Rheumatica, p. 481.) Asthma may sometimes be seen in patients subject to urticaria. The association of digestive disturbances has been spoken of under etiology.

Course and Prognosis.—The prognosis of urticaria in older children is usually favorable, and the attack is over in a few days. There is, however, in very many cases a great tendency to recurrence, and in some the disease seems to be a chronic condition, and is then extremely resistant to treatment. The urticaria papulosa of infancy and early childhood is often a most troublesome and persistent affection, resistant to treatment and prone to frequent relapses.

Diagnosis.—This is usually easy in typical cases, since there are few lesions which resemble wheals. The bites and stings of insects are to be excluded by the distribution and the history. The two may, of course, exist together. Urticaria papulosa is difficult of recognition unless the urticarial wheals chance to be discovered. If they are not, the affection may readily be supposed to be scabies; or in other cases, where vesicles predominate, may be thought to be varicella. In the latter disease the eruption is slower in appearing, the vesicles are found also on the mucous membrane of the mouth, and upon the body they dry with characteristic crusts after a few days. Scabies is distinguished by the presence of burrows.

Treatment.—Search for the cause is of great importance, especially if any disposition to recurrence is shown. As this is usually dietetic, it may be necessary in the frequently recurring cases to test the influence of the different articles of diet one after another until the offending substance is discovered. Certainly all articles should be forbidden which are known to have a tendency to produce the disease. (See Etiology.) The employment of a milk-diet is often to be recommended for cases during childhood. Attacks dependent upon cutaneous irritation need a careful investigation in this direction. Should an attack of hives have developed, the first indication is to give a freely acting purgative, preferably a saline, such as magnesia, repeated perhaps daily if the course is at all long-continued. In addition the administration of an alkali of some sort seems to be of value, such as sodium bicarbonate or potassium bitartrate, particularly if the urine is highly acid. The list of internal remedies recommended is very large. Salicylate of soda is among those in vogue, and atropine and arsenic may be tried in chronic cases. Something to quiet the nervous system and to induce sleep at night is often necessary. For this purpose may well be employed the bromides, antipyrine, chloral, or sodium-veronal. A gastric or intestinal indigestion present must be appropriately treated. Such tonic remedies as change of air constitute often the best means of cure.

Local measures are required to allay the itching. Aqueous preparations and powders are often better than ointments. A solution of sodium bicarbonate applied and allowed to dry is an old-fashioned and still popular remedy. A powder of camphor dr. 1 (3.9); zinci oxidum oz. ½ (15.5); amylum oz. ½ (15.5) frequently gives relief. Washes containing a combination of thymol and camphor-water may be applied. In older children carbolic-acid lotions may be used, but they must be employed with caution in young children and infants. A mixture of acid carbolic gr. 10 (0.65); ether. sulphuric fl. dr. 2 (7.4) and alcohol fl. dr. 6 (22.2) may be dabbed on the spots, if not too numerous, and allowed to dry, and later a dusting powder applied to exclude the air. Among other preparations recommended are liq. plumbi subacitatis (1 :10); weak vinegar; carbolized oil (1 per cent.); ichthyol ointment (1 per cent.); liq. carbonis detergens (1 :24), etc. In troublesome cases one remedy after another should be tried until that is found which gives most relief.

ANGIONEUROTIC EDEMA

(Acute Circumscribed Edema; Giant Urticaria)

Angioneurotic edema, as it was entitled by Quincke[1] is a vasomotor neurosis closely allied to urticaria and to rheumatic purpura (p. 481). It is not very common at any time of life, and least in infancy and early childhood. Writing in 1897 Newcomet and myself[2] were able to collect 14 reports from medical literature, some of more than one case, of the disease beginning in early life, with 2 instances in addition observed by ourselves. Other cases have since been reported. Doubtless it is oftener seen than has been generally supposed. It is very frequently hereditary; sometimes in several generations. The most notable instance of this is in the family reported by Osler[3] in which 25 out of 39 individuals in 5 generations suffered from the disease. The attacks may be dependent upon exposure to cold, digestive disturbances, or the action of some special articles of diet.

Symptoms.—These consist in a sudden development of localized edema which does not pit. It is situated oftenest in the face, genitals, or the extremities, although any part of the body may be involved. Sometimes the mucous membranes are affected, as of the tongue, pharynx or larynx. The attacks come on with great abruptness; the swelling is very decided; itching is generally not a troublesome symptom. The duration is generally short, a few hours or a couple of days, but sometimes much prolonged. A purpuric or erythematous eruption may be associated with the edema, and urticarial wheals may be present on other parts of the body. The disease is prone to appear in separate attacks in the same or in different regions, and sometimes at regular intervals. The affection is not a serious one except in the rare instances in which it involves the throat.

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[1] Monatssh. f. prakt. Dermat., 1882, I, 129.

[2] Med. News, 1892, Oct. 2.

[3] Amer. Jour. Med. Sci., 1888, XCV, 362.

The **treatment** consists in removing the cause as far as possible, especial attention being paid to dietetic influence and to exposure to cold. Saline laxatives and alkalies also should be given.

ERYTHEMA

Of the various forms a number seen in early life require consideration. The characteristic pathological change is a congestion produced in various ways, although in some forms the process advances beyond this and exhibits also an exudation.

Erythema Simplex.—This is one of the simplest varieties resulting from injury by the sun's rays (*erythema solare; sun-burn*); by heat applied in other ways; by friction; or by the local effect of drugs or chemical substances, such as mustard, liniments, strong soaps, acids, and the like (*erythema venenatum*). Exposure to cold produces the *erythema pernio* (chilblain; frost-bite). (See Dermatitis congelationis, p. 551.) Some of the forms of erythema simplex are the result of an action from within the body, as in cases of autointoxication, or from the ingestion of certain drugs. The latter can better be considered elsewhere (p. 549). The **symptoms** consist of simple redness in localized small or larger areas, not elevated, or possibly with some edematous swelling if the case is at all severe, but without discoverable inflammatory infiltration of the skin. A greater degree of cutaneous disturbance than this places the condition in the class of dermatitis. There is moderate swelling, burning and itching. Pressure removes the redness momentarily. The **treatment** consists in the correcting of any digestive disorder present, and especially in the administration of a purgative and the employment of a restricted diet. Locally the application of zinc-ointment, or Lassar's Paste (amylum and zinc oxide each dr. 2 (7.8); petrolatum dr. 4 (15.6)) will often give relief. Frequently, however, ointments do not agree, and the irritation is more satisfactorily treated by a powder of talc, bismuth, zinc oxide, or starch; alone or in combination; or a wash may be used, a serviceable one consisting of pulv. zinc. carb. præcip., pulv. zinc ox., pulv. amyli, glycerin., each 4 dr. (15.6); water to make 1 pint (473).

Erythema Intertrigo (*Chafing*).—This is a severer form of erythema, not infrequently passing into an eczema. It is the result of the friction between two moist surfaces, or of the continued contact of soiled diapers, particularly if there is an abnormal condition of the feces present, especially an undue acidity. The general condition of health also tends to make the skin unusually sensitive. In regions apart from the buttocks the development of the disease is favored by fatness of the baby and the occurrence of warm weather, and in older children by the taking of much exercise. The lesions are seen principally in the folds of the groins, about the genitals, the surface of the upper part of the thighs, between the nates, the folds of the neck, the region behind the ears, and the arm pits. There is redness of the skin in patches, or widely and uniformly spread, but no weeping. In severe cases burning and slight itching occur; and if neglected the skin is denuded of epithelium; papules and vesicles may form; and there is decided cutaneous swelling. The condition thus passes into eczema with a raw, weeping surface; or an infection may take place and pus-formation occur. When papules have developed the disease may strongly suggest syphilis, but is to be distinguished by the character of the remainder of the eruption in the neighborhood; the lighter color without copper tint; and the absence of any other cutaneous manifestations adjacent to the irritated area or elsewhere on the body.

The most important elements of **treatment** are cleanliness and dryness. Removal of perspiration from the folds of the neck and elsewhere will aid greatly, or the frequent changing of the diapers if the intertrigo is on the buttocks. Always after the diaper is removed the buttocks and genitals should be washed with water, or better, with starch water, dried carefully without rubbing, and dusted with a powder of talcum, bismuth, starch, stearate of zinc, or boric acid, singly or variously combined. A useful combination consists of pulv. zinc. ox., acid boric., of each, 2 dr. (7.8); pulv. talc. 4 dr. (15.6). A pledget of lint or cotton may be placed between the opposing surfaces. A calamine wash may be serviceable, such as pulv. calamini dr. 3 (11.7); pulv. zinci oxidi dr. 3 (11.7); glycerini m. XV. (0.92) alcoholis fl.dr. ½ (1.85); water to make fl.oz. 8 (237). In some cases of irritation of the buttocks a stiff paste is useful, in that it adheres better than a powder, and keeps the skin from becoming wet by the secretion. Lassar's paste (p. 545) is useful for this purpose.

Erythema Multiforme (*Erythema Exudativum*).—As its name implies, this very common disorder may appear in various forms. It differs from the two types previously described in that there is with the congestion a certain degree of inflammatory action present. The **cause** is uncertain, but it is probably a toxemia, and the disease is of the nature of an angioneurosis. It sometimes exhibits a seasonal relationship, being commoner in autumn and spring. Disorders of digestion are active in some instances. The disease may not infrequently be associated with urticaria and purpura in the purpura rheumatica previously described (p. 481), and it is certainly closely allied to this affection. Nervous influences, such as psychic, reflex, or other disturbances, may occasion it in some instances; and in others it may follow the ingestion of certain drugs (p. 549).

Symptoms.—The symptoms of erythema multiforme in the most typical cases consist in the development of bright-reddish, and later bluish or purplish, somewhat elevated patches, or of papules or vesicles, scattered or grouped, very often somewhat symmetrically arranged. The favorite seats are the dorsum of the hands, the forearms, the tibial regions and the face or neck; but sometimes the eruption is widespread. The rash may appear in one or in numerous crops. Sometimes the mucous membrane of the mouth, nose, eyelids and throat are attacked. The onset of the disease may be attended by slight fever, or rheumatic or abdominal pain, but oftener there are no constitutional symptoms, and the local ones beside the rash consist at the most of moderate itching and burning.

According to the various forms of eruption which may appear different names have been applied to the modifications of the disease. None of these are in reality distinct varieties, since several forms may sometimes be combined in the same case. In *erythema papulatum*, the most common variety, the rash consists of distinct, flat papules of a color ranging from red to purple. *Erythema tuberculatum* is similar, but with larger, more prominent lesions. *Erythema iris* (*herpes iris*) exhibits one or more areas of concentric rings of vesicopapules or papules, with color ranging from red to violet and purple; the lesions at the centre of the group fading while fresh ones develop on the periphery. *Erythema circinatum* is in the form of concentric patches, not vesicular, with the center fading while the periphery extends; and *erythema marginatum* has a large, irregular, broken, gyrate periphery, with a faded centre.

The duration of erythema multiforme is from a few days to 2 or 3 weeks. Slight desquamation and pigmentation often remain for a time. Relapses are very frequent and may prolong the course; and recurrences in succeeding years, perhaps at the same season, are not uncommon.

Diagnosis.—This is important; easy in typical cases; much less so in atypical forms. It rests upon the acute course, the multiform nature of the eruption, and the absence of severe itching. The lesions of urticaria are much more transient, are of more uniform character, have whitish centres, and itch intensely. The two diseases are not infrequently associated. Papular eczema is of longer duration, and has more severe itching and smaller and more regularly shaped papules. Erythema nodosum has a few large, hard, much raised nodules, oftenest over the front of the tibiæ and attended by burning sensations. Measles and rubella are readily diagnosed by the distribution and character of the rash and the nature of the other symptoms present.

The **treatment** consists in the giving of a purgative, the restriction of the diet, the administration of alkali, and the enforcing of rest. The salicylates are favorite remedies, of the value of which, however, I have not been convinced. Generally no local treatment is required. For the moderate itching and burning sensations present a dusting powder or a wash may be employed as in erythema intertrigo.

Erythema Scarlatiniforme.—This disorder is interesting especially from the fact that it may sometimes add to the difficulty attending the diagnosis of scarlet fever. In a previous publication[1] I have discussed the various causes more fully than the present space will permit. The occurrence of a toxemia of some sort is probably the basis of the disease. Thus a prodromal erythema may sometimes develop in varicella, variola, typhoid fever, grippe, and diphtheria, which strongly suggests scarlet fever. It may also attend the course of tonsillitis, rheumatism and sepsis. Various drugs may produce a scarlatiniform eruption in patients showing an idiosyncrasy to them, notably of those most used in childhood being quinine and belladonna, and one of the varieties of eruption seen after the injection of diphtheria-antitoxin or other serum may be a scarlatiniform erythema. The giving of an enema may occasionally be followed by the appearance of an eruption which in some cases is scarlatiniform in character. This occurs oftenest after early childhood is passed. There are exceedingly well-marked cases in which no cause whatever is discoverable.

The chief **Symptom,** as the name implies, consists of an eruption of a red color much suggesting that of scarlet fever. It may appear on any part of the body, and may be limited to this region, or may become more general. There may be some burning and itching of the skin, but slight and of brief duration, or entirely absent. With the development of the rash, or shortly before it, such mild general symptoms as chilliness, malaise, slight redness of the throat and a subfebrile temperature may be observed; but these are by no means always present, and any considerable elevation of temperature is uncommon. There may be no desquamation whatever, or it may occur after the lapse of 3 or 4 days. It is generally slight and furfuraceous. The whole duration of the attack is only a few days. An exception to all that has been said must be made as regards some of the severer cases designated *recurrent desquamative scarlatiniform erythema,* in which the eruption is widespread, intense, and more livid; the constitutional symptoms more severe; moderate fever present; and the desquamation lamellar and very abundant, in some instances the skin from the hands and feet being shed in the form of casts. The attack may last in all 3 to 4 weeks.

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[1] Medical News, 1895, Aug. 31.

There is, as indicated, a remarkable tendency to **recurrence** shown in scarlatiniform erythema, especially in the severer instances; the attacks taking place every few months or years over a long period. The **prognosis** apart from recurrence is good. The **diagnosis** is to be made especially from scarlet fever. In this connection the important characteristics of scarlatiniform erythema are the lack of contagiousness, the mildness or absence of the general symptoms in most cases, the greater tendency to limitation of the extent of the eruption, the absence of more than a slight redness of the throat and of the appearance of the tongue characteristic of scarlet fever, and the usually short duration. Yet most important of all diagnostic signs, and the only one of certain value in the severer cases, is the marked tendency to recurrence. In the first attack the diagnosis in cases of the severer class may be impossible.

Little is required in the way of **treatment.** If the scaling is very abundant and dry some emollient, such as petrolatum, can be employed. A search must be instituted for the cause.

Erythema Nodosum.—This not common variety of erythema is seen oftenest in young adults and children, especially in females. Its *cause* and nature are not entirely known. The prevailing view is that the disease is the result of a specific infection of some sort. It is sometimes seen in the course of such conditions as malaria, scarlatina, syphilis, tuberculosis and diphtheria; may follow gastrointestinal disturbances; or may be associated with rheumatism. The **lesions** are of an inflammatory nature, closely related to those of erythema multiforme, and the result of an angioneurosis, an inflammation of the lymphatics, or embolism of a cutaneous blood-vessel. Which of these is the actual method of production is undecided.

Symptoms.—The onset is generally marked by some constitutional symptoms such as fever, disturbance of digestion, and pain in and sometimes swelling of the joints. Within a few hours or a day, or without any prodromal symptoms whatever, large, nodular swellings suddenly develop, varying in number from 3 or 4 up to 2 dozen or more, and in circumference from that of a cherry to that of a hen's egg. They are of a reddish tint, turning often to bluish or purplish as they grow older, and suggesting the color of a fading bruise. They are shining, without sharply defined borders, painful and tender, and at first hard, although later they may become softer. Although they may appear upon any part of the body, they are much oftenest seen only upon the anterior tibial region upon both legs, making walking painful; less often on the arms and forearms. The constitutional symptoms of the disease generally cease after the first few days. The individual nodes last about a week, but the duration of the disease is often prolonged by the tendency for the lesions to appear in crops. Recurrence is uncommon. The **prognosis** is favorable, for although very severe and even fatal cases have been recorded, such a termination is entirely anomalous. The softening which takes place in the nodules suggests suppuration, but this never occurs.

The differential **diagnosis** is to be made from bruises, abscesses, gummata and erythema multiforme. From the last it differs in its nodular character and limited distribution. From the other conditions erythema nodosum is to be distinguished by the sudden onset, the symmetrical distribution of the lesions, their number, and the absence of suppuration.

Treatment may be limited to rest in bed and the administration of a laxative. If the pain is severe, aspirin or phenacetin may be given, or hot fomentations applied.

DRUG ERUPTIONS

Under this heading may be grouped a number of eruptions of a somewhat dissimilar nature produced by the absorption of ingested drugs. Many of these are to be classed as erythemata; others belong more properly to dermatitis of various forms. The list is a long one and only certain will be mentioned, especially those liable to occur in early life.

Antipyrine sometimes produces a macular rash much suggesting measles. Occasionally it may give rise to an urticarial, vesicular, or hemorrhagic eruption. The administration of *arsenic* may sometimes be followed by an eruption of almost any type, in some instances extensively pigmented. *Belladonna* in full doses produces an intense scarlatiniform erythema, most marked in the upper parts of the body, and especially well shown on the forehead. The *bromides* may occasion lesions of various sorts; vesicular, acne-form, pemphigoid, large papular, or echthymatous (Fig. 422). It is possible for the drug to affect the infant through the milk of the mother. *Chloral* may cause a scarlatinal eruption; sometimes one of some other nature. The *iodides* give rise oftenest to an acneform eruption, less frequently to other rashes—bullous, erythematous, purpuric, papular, or vesicular. *Opium* occasionally produces erythema or urticaria. *Quinine* in certain cases is the cause of a desquamative scarlatiniform erythema of very marked type. The *salicylates* sometimes cause erythematous or purpuric rashes. *Santonin* rarely is followed by urticaria. *Sulphonal* may bring about a rubeoloid or scarlatiniform erythema.

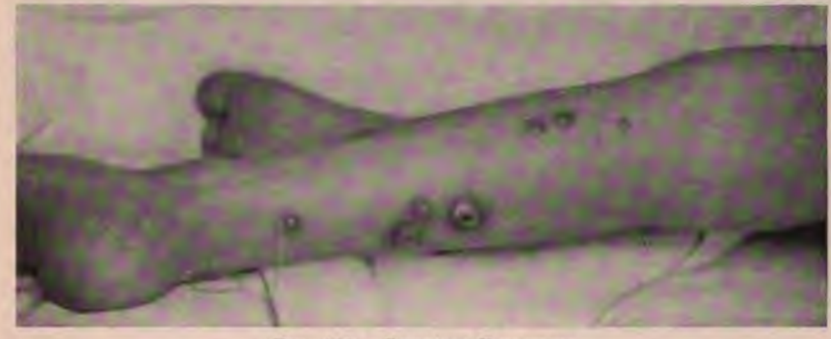

FIG. 422.—BROMIDE ERUPTION.

Ecthymatous eruption following the administration of potassium bromide in a case of cerebrospinal fever. From a patient in the Children's Hospital of Philadelphia.

Finally in this category are to be included the eruptions following the *injection of sera* (Vol. I, p. 435, Fig. 127). As seen, for instance, in that of diphtheria-antitoxin, there may be exceptionally a widespread erythema very soon after the injection is given. Generally, however, the cutaneous symptoms appear from the 5th to the 9th day after the injection, and consist oftenest of an extensive development of urticaria, or in other frequent cases of an eruption resembling erythema multiforme, or one of either a scarlatiniform or rubeoloid type. Usually a rise of temperature accompanies the appearance of the rash and articular pain and constitutional symptoms may be present. (See Vol. I, p. 464.)

DERMATITIS

All the cases grouped under the title Dermatitis are instances of inflammation of the skin. They are the result chiefly of the action of external influences. The causes are heterogenous, and the intensity of the inflammation differs greatly in the various forms. The following types may be enumerated as of interest to the student of diseases of children.

Dermatitis Venenata.—In this class are included the eruptions produced by the local effect of some substance poisonous to the skin of the individual. In many instances there exists a distinct idiosyncrasy. Most frequently in children the poison ivy (*Rhus toxicodendron*) or sometimes some other species of sumach is the cause.

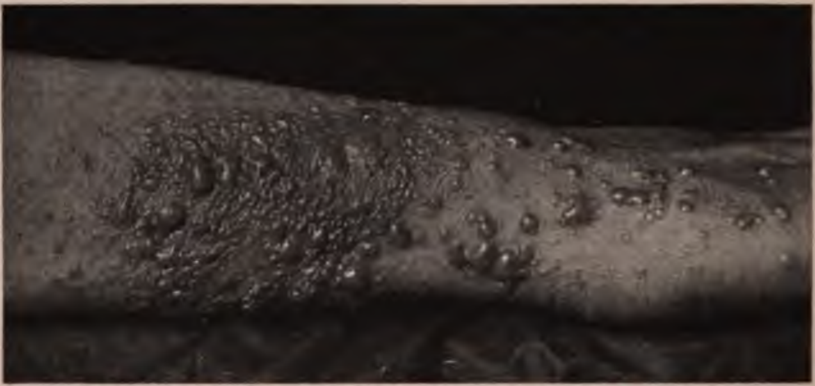

FIG. 423.—DERMATITIS VENENATA FROM EXPOSURE TO POISON-IVY.

Following shortly after exposure, vesicular and bullous lesions, not an uncommon type, hands and forearms involved, a few days' duration. (*Stelwagon, Diseases of the Skin,* 1916, 452.)

Symptoms.—These consist of burning and itching, soon followed by erythema and edema, and then by the development of numerous vesicles and bullæ, which rupture readily and discharge yellowish serum, drying in soft crusts (Fig. 423). The most common situations are those most exposed to direct contact with the poison, or through transmission by the hands; viz. the hands, face, and external genitals. Not infrequently the swelling of the face is so great that the eyes are almost or quite closed. A slight febrile reaction may be present, and the general discomfort is often intense. At the end of about a week the acute symptoms are usually over.

Of the numerous other plants which are productive of a dermatitis in susceptible persons, the primrose (*primula*) stands next in frequency. The eruption may be like that of the poison-ivy rash, or be bullous or urticarial in nature.

Among drugs or other substances used locally and capable of giving rise to dermatitis venenata may be mentioned croton oil and tartar emetic, either of which produces a pustular eruption; iodoform, causing an erythematous or vesicular dermatitis, and carbolic acid occasioning an erythematous eruption followed in severe cases by actual loss of substance, as in a burn. I have seen 2 cases of this accident in children in whom a proprietary lotion containing carbolic acid had been applied to the face according to the directions given upon the bottle. Iodoform not infrequently causes an erythematous dermatitis, and mercurial ointments may sometimes produce an inflammation of a severe nature.

The **prognosis** in dermatitis venenata from any cause is usually good, although sometimes an eczema may follow as a sequel. The **diagnosis** from eczema is not always easy. Dermatitis venenata is distinguished especially by the very acute onset, the severity of the lesions, and the limitation to regions where there is a history of the possible action of some locally toxic substance. The **treatment** will be considered especially as it applies to the commonest form, poison-ivy rash. Other varieties are benefited by the same applications. A large number of remedies have been suggested. The employment of lotio nigra followed by zinc ointment has long been a popular remedy, as has that of diluted liquor plumbi subacetatis, or a saturated solution of boric acid. With all these it is necessary to keep the surface of the skin constantly moist by the wearing of a mask; otherwise when dry the skin feels more tense and inflamed than ever. The addition of a little glycerine to the lotion may prevent this. In other cases the application of an oily substance often gives greater relief. For this purpose petrolatum alone may be used, or with 2 gr. (0.13) of menthol or 5 or 10 gr. (0.32 or 0.65) of carbolic acid to the ounce (31). The addition of 2 dr. (7.8) of bismuth subcarbonate or of zinc oxide is sometimes useful. Carbolic acid should, however, not be used in the case of infants, on account of the danger of absorption. For the cases of dermatitis venenata in which the eruption is chiefly erythematous powders may be of benefit, such as zinc oxide, boric acid, bismuth, or talc alone or in combination.

Dermatitis Actinea.—This is merely the condition of sun-burn in which the lesion has passed beyond that of a simple erythema. The parts are more swollen and vesicles and bullæ appear. The same treatment may be applied as in dermatitis venenata.

Dermatitis Congelationis (*Chilblain; Frost-bite*).—The severe second and third degrees of this disorder are entirely in the domain of surgery. In the milder first degree the condition depends upon exposure to moderate cold, often combined with a state of lowered general resistance or an individual predisposition. The **lesions** consist in the development of circumscribed burning and itching areas of a dusky-red color, hard, and somewhat elevated. They occur oftenest upon the hands and feet, the ears, cheeks, and nose. They may last for days or longer; and may then disappear entirely, or return whenever the parts are exposed to cold or to heat. Chilblains once formed are liable to persist in this way during the whole winter, and in those predisposed to return in the autumn with the beginning of cold weather. Sometimes if much irritated, as by the friction of a shoe, blebs may form upon them, or ulceration take place. The **prognosis** is usually good, but sometimes the disorder continues to appear in the winter-time year after year.

In the **treatment** of the initial lesion, the frozen part should be rubbed with snow or cold water until the circulation is well established, and the patient kept away from the heat. Later there should be efforts made to prevent recurrence by the wearing of warm clothing, especially woollen stockings and gloves, and the protection of the ears against cold.

In the chronically recurring cases care should be taken to improve the general health; and locally in such cases benefit sometimes follows painting the chilblains with diluted tincture of iodine, or a mixture of tincture of iodine fl.dr. 1 (3.7), flexible collodion fl.oz. 1 (30). Should bullæ have formed, these should be opened, if not already broken, and the parts dressed with carbolized petrolatum (gr. 10 (0.65):oz. 1 (31)) or an ichthyol ointment (ichthyol dr. ½ (1.9), petrolatum oz. 1 (31)).

Dermatitis Gangrenosa.—The rare disease going by this name develops oftenest in debilitated infants, especially those recovering from varicella. (See Varicella Gangrenosa, Vol. I, p. 387, Fig. 91). In other instances it may arise without other previous cutaneous disorder. The direct cause is microbic, various pyogenic germs seeming to be active factors. Among these is the bacillus pyocyaneus, as I have seen in 1 instance. The **lesions** consist of multiple areas of cutaneous gangrene. They develop as vesicles or papules, and terminate in necrotic ulcers, often hemorrhagic, and of varying size. Some are large and of irregular shape from confluence of several lesions; others form smaller oval or round, punched-out ulcers. They are few or numerous, and usually continue to appear in successive crops during several days or a week. Constitutional **symptoms** may be absent in the mildest cases, but oftener they are severely septic, and in the majority of instances death from sepsis occurs. The **diagnosis** is readily made; resting upon the multiple gangrenous destruction of tissue and the severe course of the disease. **Treatment** is principally strongly tonic and supportive, as in other septic conditions. Locally it must be carefully antiseptic.

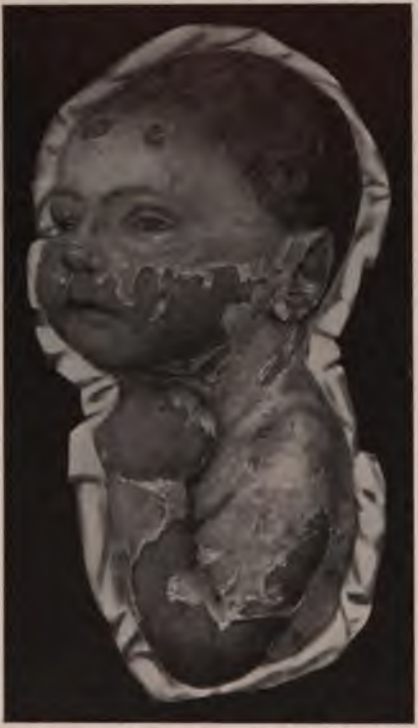

FIG. 424.—DERMATITIS EXFOLIATIVA NEONATORUM.

Occurring in a 12-day old child, ill 1 week. Died on the 13th day. (*Photograph after colored plate by von Reuss, Die Krankheiten des Neugeborenen*, 1914, 363, *Fig.* 70.)

Dermatitis Exfoliativa Neonatorum (*Ritter's Disease*).—This affection was first described by Ritter[1] who reported upon 297 cases occurring in infants up to the age of 5 weeks. In spite of this large number the disease in the experience of most physicians is decidedly uncommon. Its cause is not known, and it is uncertain whether it is of a toxic or of a septic nature, or an exaggeration of the physiological desquamative process. It has been considered to be closely related to pemphigus, or to be a variety of it (Knöpfelmacher and Leiner)[2] and Tamm[3] advanced evidence to prove its identity with impetigo contagiosa. It has been observed chiefly in asylums for infants, and has occurred epidemically.

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[1] Centralz. f., Kinderh., 1878, No. 1. Archiv. f. Kinderh., 1880, 1, 53.

[2] Jahrb. f. Kinderh., 1904, LX, 178.

[3] Dermatol. Zeitschr., 1914, XXI, 670.

Symptoms.—After the normal desquamation of the skin following birth is over, but rarely before the end of the 1st week, the disorder begins acutely as an intensely red area, oftenest about the mouth, with the formation of fissures here. This redness rapidly increases in degree and spreads until the whole body is covered. Meanwhile the skin is thickened and edematous, and the epithelium separated in large pieces, the surface beneath often being moist and looking as though scalded (Fig. 424). There are sometimes a few bullæ present; but the surface of the body in general is dry. The mucous membrane of the mouth, nose, and conjunctiva may exhibit erosions. The pathological findings are uncharacteristic. Constitutional symptoms, including fever, are generally absent. The duration of the disease is 1 to 2 weeks in favorable cases; but in others it may be lengthened by continued desquamation of the skin.

The **prognosis** is grave. About 50 per cent. of the cases die from debility attending the prolongation of the process, or from the occurrence of some complication, as gastrointestinal disorders, pneumonia, or purulent invasion of the skin or umbilicus. The **diagnosis** rests upon the combination of fissures about the mouth, with very abundant desquamation of the skin in large pieces over the body. The fissuring is distinguished from that of hereditary syphilis by the absence of other evidences of that disease. The chief condition to be separated from it is the widespread macular, papular, or chiefly squamous erythema which is sometimes seen in the new born, and which is an exaggeration of the normal process, or an instance of toxic or septic erythema in weakly new-born infants. It is not attended by fissuring. Erythrodermia desquamativa is also to be distinguished. (See below.) The constitutional **treatment** is supportive, and the local consists in protecting the skin and keeping it soft with petrolatum, zinc ointment, or Lassar's paste (p. 545).

ERYTHRODERMIA DESQUAMATIVA

(Leiner's Disease)

The first detailed description of this affection, probably not uncommon, was by Leiner[1] who reported upon 43 cases. It appears in early infancy at the end of the 1st or the 2d month of life, and begins as a thick seborrheic crust over the scalp, or in the form of erythematous spots on the trunk. In well-developed cases the whole cutaneous surface becomes intensely red and covered with large yellow or whitish scales which are [readily removable, leaving the surface dry beneath them. The nails are involved. The infant exhibits as a rule no fever or other constitutional disturbance, except a loss of weight and the uniform presence of diarrhea. There is no itching. Histologically the disease is of the nature of a universal dermatitis; not a seborrheic eczema. The **cause** would appear to be an autointoxication of some sort, dependent upon the nature of the food. In all but 2 of Leiner's cases the infants were breast-fed, as were 10 also of the 16 cases reported by Beck;[2] and improvement did not begin until the diet was changed. Breast-feeding is not, however, an essential etiological factor, since of Moro's[3]

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[1] Brit. Jour. Child. Dis., 1908, V, 244. Arch. f. Dermatol. u. Syph., 1908, LXXXIX, 65.

[2] Arch. f. Dermatol. u. Syph., 1911, CVI, 9.

[3] Münch. med. Woch., 1911, LVIII, 499.

11 cases only 4 were breast-fed. The development of the disease is favored by the existence of an unusual degree of the physiological desquamation of the skin after birth.

The **prognosis** is usually favorable and recovery takes place in a few weeks. The results are not always so good, since 15 of Leiner's patients died. In the series of Moro and of Beck recovery occurred in every instance.

The **diagnosis** is to be made especially from the dermatitis exfoliativa of Ritter. The latter affection begins around the mouth; there is often moist exudation under the epidermis, and the tissues beneath this exhibit degenerative changes. There is no seborrhea of the head; the onset and course are more acute. Eczema is distinguished by the presence of itching papules.

The **treatment** is chiefly dietetic. In addition the patient may receive bran baths and be smeared with petrolatum.

MILIARIA

(Prickly Heat)

Etiology.—The cause of this exceedingly common disorder is an excessive energy of the sweat glands brought about generally through the influence of heat. It may be seen in the course of the acute febrile diseases, especially typhoid fever, rheumatism and scarlatina (see Scarlatina miliaris, Vol. I, p. 315), but may also occur in debilitated states attended by free perspiration. Prominent causes are exposure to hot summer weather, the wearing of too warm clothing, and hard exercise.

Symptoms.—The disease may be divided into several forms. In the first, *sudamina*, or *miliaria crystallina*, there is merely a disordered action of the sweat glands, with obstruction to the exit of the liquid; and as a result large numbers of discrete, usually closely placed, minute, transparent vesicles with clear contents develop principally upon the trunk. There is no inflammatory element present, and, strictly speaking, according to the classifications adopted by many, this condition should not be called miliaria, inasmuch as in the latter inflammation is either a primary or a secondary development. Sudamina is the form seen especially in fevers or debilitated states. There are no other symptoms, and the lesions soon dry, but relapse readily occurs.

Miliaria of the inflammatory type may be divided into several subvarieties. *Miliaria vesiculosa* is the term applied to the form in which the numerous vesicles are situated upon a red base. The crowding of the vesicles is often so great that the whole of the affected skin may have a red appearance, and the title *miliaria rubra* is then employed. If the basal congestion is slight, or has faded, and the vesicles become less translucent, the term *miliaria alba* is used. Still another form is *miliaria papulosa* (*lichen tropicus*) in which the production of very numerous crowded, minute papules is the predominating feature. Tiny vesicles may occupy the summit of the papules; and vesicles are in addition interspersed among these vesicopapules.

There is no sharp distinction between these forms of inflammatory miliaria, and generally there is a commingling of the papular and the vesicular lesions, the one form predominating in one case and the other in another.

The onset of miliaria of the inflammatory type is sudden and the eru[illegible]sually rather widely spread. The itching and burning may be slight, but are often intense. The rash lasts a few days, or may be continued almost indefinitely by the appearance of new crops unless the cause is removed. As recovery takes place the papules disappear, the vesicles dry without rupture, and a slight, fine desquamation follows, if the vesicular element has been a pronounced one. In severe or long-continued cases, especially where there has been much scratching, eczema or secondary pustular lesions may develop.

Diagnosis.—The disease is to be distinguished chiefly from eczema. In the latter, however, the operative cause of miliaria is absent, the onset is less sudden, the lesions are larger, and rupture of the vesicles with oozing occurs. I have seen cases of well-marked scarlatina miliaris in which the vesicles were so numerous that the appearance of the skin beneath was obscured, and a mistaken diagnosis might easily have been made and the infectious nature overlooked, if other symptoms than the cutaneous had not been considered.

Treatment.—The removal of the cause is the first requirement. Many children, and especially infants, are dressed far too warmly, and even in the winter season readily develop miliaria on this account. The skin of some infants is intolerant to wool even in winter time, and in the summer, in the hot or even the temperate zone, it is frequently necessary in the case of many others to employ undergarments of some other nature. (See Vol. I, p. 71.) Another important preventive measure is the ensuring of cleanliness by frequent bathing. Keeping the skin as dry as possible by the application of an unirritating powder, such as unperfumed talcum, should also be practiced. Should an attack develop, the same measures are to be employed with even greater thoroughness. In addition lotions or dusting powders may be used. Among the former is to be recommended starch water; a solution of bicarbonate of soda (1:200); a calamine lotion (calamine; zinc oxide each dr. 2 (7.8); water fl.oz. 4 (118)); or lotio nigra. Among powders may be mentioned lycopodium; equal parts of oxide of zinc and starch; and combinations of bismuth, boric acid and zinc oxide. A camphor, zinc and starch powder (p. 544) is often very efficacious. A laxative may be administered with advantage, and the diet made lighter than usual.

SEBORRHEA

Etiology.—This disorder consists in an abnormal increase in the secretion of the sebaceous glands. Attacking the scalp it is of very frequent occurrence in early life and particularly in infancy. The cause of the disease of the glands is not certainly known, but the view that it is dependent upon the action of bacteria is becoming prevalent. Probably, too, debility, anemia, indigestion, and the like predispose to its development.

Symptoms.—The disease is commonly divided into *seborrhea oleosa* and *seborrhea sicca*, according as oiliness or dryness predominates in the secretion. The first of these is seen in older children approaching puberty, producing an oily state of the nose and forehead, the front of the chest, and the scalp. The second is the condition to which especial attention is given here. It is common particularly in infants. The deposit is composed of a mixture of the seborrheal secretion with epithelial cells and dirt; and forms a thick, oily, yellowish crust (*milk-crust*). It is usually most marked on the vertex, but if neglected may cover the greater portion of the scalp and even sometimes attack the forehead, nose, and chest. Removal of the crusts shows the skin in an apparently normal state; but not infrequently, if the case has existed for some time, the underlying skin has become red and infiltrated through the development of an eczema. The removal of the deposit is easy, but the final cure is often only slowly attained, since relapse occurs readily. In older children the deposit on the scalp is usually in the form of small, whitish scales (*dandruff*).

The **prognosis** is on the whole good, although the course may be long-continued in obstinate cases, and there is a decided disposition to recurrence. In the cases of dandruff of older children there is a tendency to loss of hair. The **diagnosis** is easy when the oily character predominates. The crusted and scaly condition, especially on the scalp, is to be differentiated from eczema by the absence of all evidence of inflammation. Should this symptom be present the disease is to be regarded as seborrheic eczema.

Treatment.—In the treatment of milk-crust, the first step is that of softening the mass by the application of a bland oil or of petrolatum, and then of dislodging it by washing with hot soapy water. Several courses of treatment of this sort may be necessary to accomplish complete removal. All violence, such as by the use of a fine-tooth comb, is to be avoided. After the cleansing, the application of a stimulating ointment is required, such as of resorcin (gr. 10 (0.65), petrolatum oz. 1 (31)); salicylic acid (gr. 10 (0.65), petrolatum oz. 1 (31)); or precipitated sulphur (dr. 1 (3.9), petrolatum oz. 1 (31)). The washing process should be repeated as soon as any tendency to fresh crusts is seen. In older children with dandruff or with the oily form of seborrhea, lotions of resorcin, carbolic acid or sulphur may be preferable. The employment of one of the superfatted medicated soaps is of value, containing resorcin, sulphur, or salicylic acid, alone or in combination.

PSORIASIS

Etiology.—This is a not uncommon affection of the skin, of which about half of the cases begin in childhood. Nielsen[1] in an analysis of 520 cases found 44 per cent. developing under the age of 15 years, few of them, however, being under 5 years. Yet cases in the 1st month of life have been reported. Davis[2] recorded an instance in an infant of 6 weeks, and collected from medical literature a number of others developing in the 1st year of life. In a case reported by Rille[3] the eruption was discovered on the 5th or 6th day of life. A familial tendency has occasionally been observed. The disease is commonest in winter, and a patient may be free from it in the summer time, to suffer a recurrence with the return of cold weather. It has been supposed to be neuropathic, and by many to be parasitic, but nothing certain is known of it in these respects.

Pathological Anatomy.—The lesions consist of a chronic inflammation of the papillary layer of the skin. The rete Malpighii is thickened and shows proliferation, with dilatation of the vessels, edema, and cellular infiltration. The papillæ are much elongated. There is an accumulation of the horny epithelial cells which should have been desquamated.

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[1] Monatsschr. f. prakt. Dermatolog., 1892, XV, 325.
[2] Brit. Jour. Child. Dis., 1914, XI, 22.
[3] Wien. med. Woch., 1895, XLV, 2097.

Symptoms.—The eruption begins as more or less rounded spots, pin-head sized, which extend at the periphery until they may attain an area of a rounded or irregular form and of an inch (2.5 cm.) or more in diameter. At the beginning the spots may be smooth and red, but they soon become covered with a crust of silvery scales. They are always very sharply circumscribed, the transition to the surrounding healthy skin being abrupt. Gentle scraping of the lesions easily removes the scales, and reveals a red, dry base upon which numerous bleeding points develop if the scraping is continued. Often the centre of the larger areas shows a tendency to recovery, thus leaving the outer scaling border in the form of a ring. The spots may be few in number or widely spread. They involve by preference the extensor surfaces of the limbs, especially about the joints, the trunk and the scalp. The nails may sometimes be brittle and opaque or pitted. The face, palms and soles are not often attacked. Itching and other subjective manifestations are entirely absent or insignificant.

Course and Prognosis.—The disease is essentially a chronic one, new lesions continuing to develop slowly or rapidly during an indefinite period. Although yielding to treatment, or disappearing of itself, recurrence of the affection is almost sure to take place, perhaps after months or even years. The prognosis for recovery from the individual attack is good.

Diagnosis.—Ordinarily this is readily made. In children, especially in the first attack, the lesions are often small, smooth, and do not show the characteristic silvery scaling until gentle scraping is employed. Eczema itches severely, has a less sharply circumscribed border, and any crusting present is yellowish, or, in the case of seborrheic eczema, greasy.

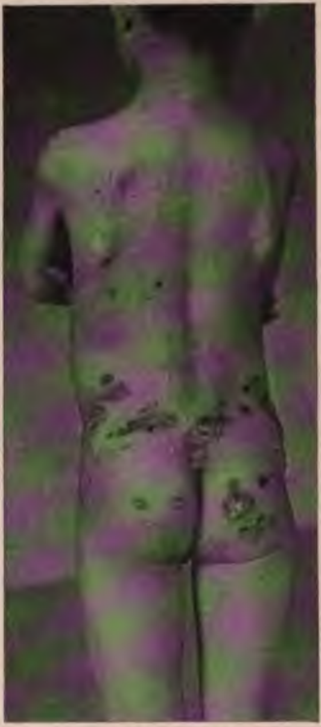

FIG. 425.—PSORIASIS.
From a patient in the Children's Hospital of Philadelphia.

Treatment.—Internally one of the most favorite remedies is arsenic. Salicylates are also recommended and alteration of the diet may be tried. But inasmuch as the cause is not known, internal medication is empirical and uncertain in its results. Removal to a warm climate may serve to cure an attack. External treatment is of more importance. Many remedies have been employed, concerning which reference must be made to treatises upon dermatology. Among some which have been extensively used are preparations of salicylic acid; sulphur; ammoniated mercury; tar in some form; chrysarobin; and pyrogallol. The last two are often decidedly irritating, especially in children, and others may well be tried first. A warm bath should precede the application of the medicament.

ECZEMA

Etiology.—Eczema, one of the most common and often most troublesome of the diseases of early life is dependent upon more than one cause. These causes may be arranged into (1) constitutional and (2) local.

Of *constitutional* causes one of the most prominent predisposing influences, especially in infancy, is a sensitive character of the skin. This is without doubt inherited in many instances, and accounts for a family predisposition often seen. In very susceptible individuals the most trifling exciting cause may be sufficient to produce an attack. There appears to be an inherited tendency in families subject to rheumatism and gout. Disturbances of the urinary secretion may occasion the disease, as when the urine is scanty, or contains albumin, sugar, or an excess of uric acid. There is also powerfully active the influence of the exudative diathesis, and as a consequence eczema in infancy may be replaced by asthmatic attacks later. Various gastrointestinal disturbances give rise to attacks, constipation being prominent among these; and certain articles of food appear to produce eczema in certain children, without any evidence of indigestion being present. It is difficult to determine in advance what such articles may be. Thus in one infant under my observation the change from an oatmeal water to a barley diluent in the milk was attended by prompt and permanent cure; and in another the same result followed a change from a maltose to a lactose addition. That sugar of any sort is a factor in some instances seems certain, and in many cases an excess of fat appears to be operative; while Finkelstein[1] and others have assigned the evil results to the salts of the milk. While it is not proven that the proteins possess any influence in determining an attack of eczema, yet it has been shown by Schloss[2] that the subjects of this disease will in many instances exhibit a cutaneous reaction to certain proteins. From these statements it is evident, then, that whereas the influence of the diet is undoubtedly great, the causative element would appear to vary with the case, and that over-feeding of any kind is very prone to produce the disease. It is a matter of common observation that very well nourished, breast-fed or bottle-fed babies are much more prone to eczema than are the underfed atrophic infants.

The local causes of irritation, although important, are so to a lesser degree particularly in infants. The influence of bacteria has been much discussed, but the weight of dermatological opinion is against the theory of eczema being primarily dependent upon germs of this nature. In seborrheic eczema the seborrhea is probably parasitic and the eczema a complication; while the production of pustular eczema by the action of pyogenic organisms is also a secondary process.

Certainly irritation of some sort is the chief local factor. This may depend upon uncleanliness, irritating discharge from the nose or ear; the prolonged contact of wet diapers; the presence of pediculi or of scabies; exposure to cold or to high winds; irritating clothing, especially woolen, or garments containing anilin dyes; the local action of various drugs, among them formaldehyde, sulphur, iodoform, etc. Eczema may follow miliaria, dermatitis, intertrigo, or especially seborrhea, or result from the use of strong alkaline soaps, or even too frequent bathing in simple water.

Pathological Anatomy.—The disease is a catarrhal inflammation of the skin, with an exudate which is either fluid or plastic. It begins with hyperemia of the vessels followed by proliferation of the epithelium and connective tissue of the papillary layer, together with edema and cellular infiltration here and in the corium. Exudation of serum loosens the epithelial cells, and infiltration by leucocytes produces vesicles or papules. Rupture of the vesicles occurs and crusts form. Pustules are not in themselves a part of the process, but the result of a secondary pyogenic infection.

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[1] Med. Klinik, 1907, III, 1098.

[2] Trans. Amer. Ped. Soc., 1915, XXVII, 62.

Symptoms.—Numerous classifications of the forms of the disease have been made, depending upon the characteristics of the various lesions, their situation, their duration, and the like. Any such classification cannot well be applied to early life, since lesions of different types may occur together in the same patient. We may conveniently divide the symptoms into (1) those seen in infants, oftenest in the 1st year, and (2) those occurring in older children.

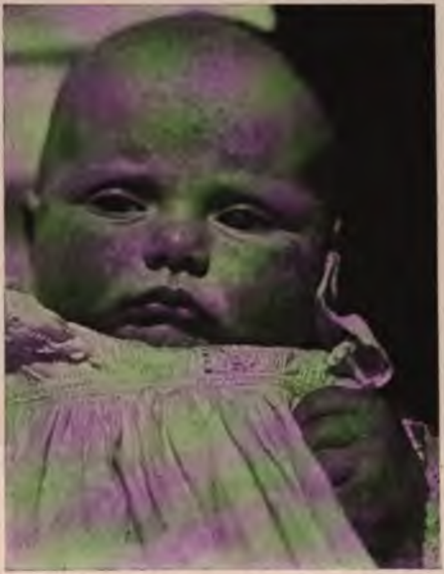

FIG. 426.—ERYTHEMATO-VESICULAR ECZEMA OF THE FACE.
Courtesy of Dr. M. B. Hartzell.

1. Eczema in Infancy.—The situation in infants is oftenest the face or head, which may be partially or completely covered by the eruption (Figs. 426 and 427). The favorite regions next to the head are the genitals, the folds of the joints, breast, abdomen and back. Much less often the eczema is nearly universal in distribution. In typical cases the disease begins as an erythematous or papular redness and roughness, with severe itching; but the affected area soon becomes moist, and close inspection shows a multitude of vesicles, many of them ruptured and discharging a transparent, yellowish, sticky fluid. Often the whole area exhibits a swollen, raw, weeping surface, rendered visible only after the removal of the thick, yellowish crusts which form upon it. Secondary purulent infection may cause the production of pustules or the discharge of pus from the raw surfaces. The tendency to pustulation is much the greatest in early life. The extreme itching results in scratching, which is followed by effusion of blood. The skin is much swollen, and the neighboring lymph-glands become enlarged and may suppurate.

The constitutional symptoms vary with the severity of the attack. The milder cases suffer but little if at all, but in others the sleepless days and nights and the constant crying produced by the itching result in distinct loss of health and impairment of nutrition. In the worst cases there may be fever and severe nervous symptoms.

Not all cases exhibit these typical manifestations. In many instances the eczema is of a less acute character. The skin then becomes dry or nearly so, red, swollen, scaling, and cracked. There is severe itching as in the moist type. This form is oftener seen on the trunk and extremities than on the face. The two conditions may appear at different times in the same locality; the more acute stage being marked by vesiculation; the less acute by the drier condition.

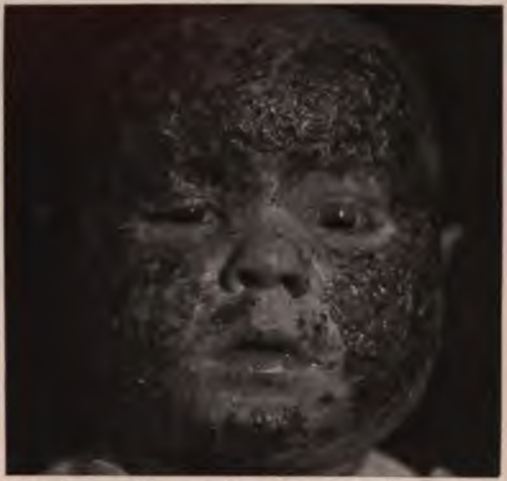

FIG. 427.—SEVERE CASE OF ECZEMA OF THE FACE AND SCALP.
(*Stelwagon, Essentials of Diseases of the Skin*, 1907, 105.)

2. Eczema in Older Children.—After the period of infancy, eczema approaches the adult type in proportion as the age increases. Certain varieties are of especial interest. There is to be noted the eczema of the eyelids and of the nostrils and upper lip common in children with tuberculous cervical glands ("scrofulous eczema"). There is also at this time of life less tendency to involve the face; and the flexures of the joints are favorite seats. The scalp, too, especially the occipital region, is often affected from the irritation set up by pediculi, or by the persistent scratching which these engender. Independently of the presence of pediculi there is frequently seen a pustular eczema of the scalp characterized by the development of few or many crusts, sometimes fused and with the hair embedded in them. The neighboring lymphatic glands are enlarged. In still other cases eczema tends to be of a papular type, with intense itching.

Course and Prognosis.—It is in most instances easy to relieve an acute attack of eczema, either completely or in part, but there is a great tendency to relapse, and permanent cure is very difficult. On the other hand, some cases appear absolutely resistant to any treatment employed. It is fortunate, however, that after the 1st year of life eczema has a tendency to disappear of itself when a child is put upon a diet other than one solely of milk. On the whole the prognosis for complete recovery from the disease is unfavorable during the 1st year; and in the eczema developing or continuing after this period the prevention of recurrences is often very difficult. As far as life is concerned the prognosis is favorable. Very severe cases may, however, exhibit fever and nervous symptoms and develop a toxic state which may prove fatal. There are instances in which nephritis occurs, and others in which sudden death has taken place, doubtless due to the presence of the status lymphaticus.

Diagnosis.—This is generally easy; sometimes difficult. It rests upon the redness, infiltration, swelling, and, in the acute cases, the oozing of the skin; the severe itching; and the chronic course. The disease is distinguished from *erythema* by the evidence of cutaneous infiltration; from severe forms of *dermatitis*, as that which poison-ivy produces, by the less violent and sudden onset, the longer course, the localization, and the absence of specific cause; from seborrhea by the presence of a red inflamed area under the crusts; from *miliaria* by the slower onset and the tendency to rupture of the vesicles. *Scabies* resembles eczema in the severity of the itching, but it is most frequent in special regions, principally the flexor surfaces, the arm-pits, the hands, and between the fingers or the toes. The lesions are discrete. Careful examination will reveal burrows; and other members of the family are likely to have the disease. Eczema frequently is combined with scabies. *Erysipelas*, especially of the face, suggests eczema, but there is greater swelling, less uniform fine vesiculation, and the presence of fever. *Impetigo* may resemble pustular eczema, but is distinguished by the fact that the crusts are produced by separate isolated pustules, not by a pus-secreting surface. *Syphilis* may simulate a papulosquamous eczema, but its lesions are larger, more discrete, less scaly, and of a darker red or often a coppery tint, and do not itch. Other symptoms of the disease are generally present.

Treatment. **Constitutional.**—In determining the character of this the nature of the cause and of the lesions must be taken into consideration. The digestive condition must be studied with especial care, such foods excluded as seem possible factors in producing the disease, over-feeding avoided, and any tendency to constipation relieved. In breast-fed babies an analysis of the milk should be made to determine whether any ingredient is present in excess. In thriving artificially fed babies the same consideration should be given, and the question determined whether the food is too strong in some particular element or too abundant in quantity. A great many infants with eczema are over-fed. In older children some radical change in the diet may be necessary. The total amount of food should be reduced and a trial may be made of the omission of eggs, milk, or sugar, or the diminution of the amount of starchy food. Naturally care must be taken at any age that the alteration of the diet is not such as will impair the general nutrition. Based upon the frequency with which a cutaneous reaction to proteins occurs in eczema, Blackfan[1] tried with success in older children the removal of all or some of the animal protein from the diet.

In some cases tonic remedies are indicated, even though the children appear to be well-nourished. Some are anemic and flabby and will be benefited by cod-liver oil or iron. In obstinate cases arsenic has had a reputation greater formerly than now. It has seemed to me, however, of undoubted benefit in certain instances. It is questionable whether it possesses any specific properties. Based on the association in some older children of eczema with asthma, recurrent bronchitis, or recurrent vomiting, the continued administration of an alkali is to be recommended, bicarbonate of soda in full doses being serviceable for this purpose. Free action of the kidneys is to be maintained by the ingestion of plenty of water and the administration of potassium citrate or potassium acetate. One of the most important forms of internal medication is the use of purgatives. In infancy a course of calomel and soda or other purgative may be given every 5 or 6 days, and is sometimes very efficacious. To older children the same may be administered, or citrate of magnesia employed. Pains must be taken that on intermediate days constipation is avoided by the administration of some gentle laxative, such as rhubarb and magnesium sulphate; senna and cascara; milk of magnesia; phenolphthalein, and the like. (See Constipation, Vol. I, p. 760.)

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[1] Amer. Jour. Dis. Child., 1916, XI, 441.

Local Treatment.—This is of great importance. The removal of all local causes is to be secured, such as irritation by the clothing, exposure to cold and wind, or excessive perspiration. Scratching must be prevented. To accomplish this some mechanical restraint is often necessary. Pasteboard splints bound around the elbows are very useful in preventing the scratching of the face, although the child can still accomplish his purpose to some extent by rubbing with the upper part of the arm. Serviceable, globular, aluminum mitts (Fig. 428) are on the market, furnished with a textile cuff to tie around the wrist. I have found these of great service. In eczema of the buttocks in infancy care should be taken that the diapers are not washed with soda, or ever used a second time after merely drying of the urine. Before a local treatment of any part is given, the region should be carefully freed of crusts; but the employment of soap and water, or even sometimes of plain water for this purpose or for bathing, is frequently contra-indicated. Starch water or a solution of boric acid is to be preferred; or some oily substance such as petrolatum or cold cream may be used instead. The choice of applications is large, and must be made according to the condition of the skin and the duration of the disease. When the eczema is in its most acute stage the applications should all be of a sedative nature. Some cases are most benefited by ointments or pastes, the latter being less suited for the scalp on account of the adhesion to the hair. Among those suitable is one of acid boric gr. 20 (1.3), ung. zinci oxidi oz. 1 (31); bismuth subnitrate dr. 1 (3.9), ung. aquæ rosæ oz. 1 (31); or Lassar's paste (p. 545). Any of these may have the addition of menthol 1 to 2 gr. (0.065 to 0.13) to relieve the itching; or in older children phenol, gr. 5 (0.32), if the case is not too acute. Whatever dressing is applied must be in sufficient quantity and frequently enough renewed to keep in constant contact with the part. The pastes have an advantage in this respect, in that they are thicker and more adherent. Upon the face it is best to have the remedy spread upon a linen mask. Elsewhere it can be applied thickly upon a soft cloth, and secured with bandages. Little good is accomplished by merely smearing the application upon the body and allowing it to rub off promptly on the clothing. Some cases do not tolerate oils well, and for these lotions are to be preferred. Here there is a choice, among others, of a saturated solution of boric acid; equal parts

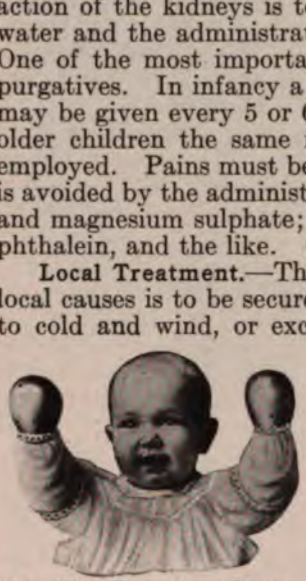

FIG. 428.—THE HAND-I-HOLD BABE MITTS APPLIED.
Courtesy of R. M. Clark and Co. Newton Centre, Mass.

of lotio nigra and lime water; or one of bismuth subcarbonate dr. ½ (1.9), water fl.oz. 1 (30); or calamine gr. 20 (1.3), zinc oxide gr. 30 (1.9), aqua rosæ fl.oz. 1 (30). Aqua camphoræ may be used as a diluent in order to relieve the itching, with 10 minims (0.62) of glycerine added to prevent too rapid drying. The affected part should be kept constantly moist by wetting cloths with the lotion, bandaging them upon the diseased areas, and renewing frequently. If a face-mask is objected to by the family, or if the region is one where it is impossible to use bandages, the medicament may be applied in the form of a mucilage and allowed to dry on; such, for instance, as Pick's formula[1] (tragacanth 5, glycerine 2, boiling water 100) to which the drug selected may be added. The addition of 2 per cent. of boric acid aids in preserving it. A superior preparation is the bassorin formula of Elliot.[2] (Bassorin 48, dextrin 25, glycerine 10, water to make 100) which can be similarly medicated. To any of the lotions small amounts of carbolic acid can be added if the itching is intense. In many cases of vesicular eczema with free oozing, powders are still better. The substances already mentioned may be employed; or such combinations, for instance, as of thymol gr. ¼, (0.016), acid boric dr. ½ (1.9), powdered talc oz. 1 (31); or of camphor dr. 1 (3.9), amylum oz. ½ (15.5), zinc oxide oz. ½ (15.5); or bismuth subcarbonate gr. 20 (1.3), talc oz. ½ (15.5); amylum oz. ½ (15.5).

For the less acute or more prolonged cases of vesicular eczema, as for those rather of a papular or squamous form, more stimulating treatment is often needed. Here the preparations of tar constitute one of the most efficacious applications. We may use ung. picis liquidis dr. 2 to 4 (7.8 to 15.6), ung. zinci oxidi oz. 1 (31); or the remedy may be employed with great satisfaction in the following: oil of cade fl.dr. ½ (1.9), flexible collodion fl.oz. 1 (30). The mixture may be painted over the diseased region and allowed to dry. It does not need to be removed, but fresh should be applied as it peels. I have had excellent results with this. In other cases tar may be applied as a lotion (coal-tar 1, tincture of soapbark 6), or may be mixed with Lassar's paste (p. 545). In the pustular cases one of the best remedies is the combination of hydrargyrum ammoniatum grains 10 to 20 (0.65 to 1.3), lanolin and petrolatum each oz. ½ (15.5).

HERPES SIMPLEX

(Fever Blisters)

The nature of this disease is not clearly understood. It may perhaps, like herpes zoster, be connected with a disorder of the nerves. The **causes** are various, among them being any slight febrile disturbance; cold in the head; indigestion; exposure to cold winds; or excess sunshine. Often no cause whatever can be discovered. With certain diseases there is a special tendency for herpes to develop, notably here being croupous pneumonia, cerebrospinal fever, and malaria. It is rare in typhoid fever, but I have occasionally observed it.

As ordinarily seen the **symptoms** manifest themselves as vesicles of pin-head or slightly larger size, closely approximated in a small group, and situated upon a slightly reddened base and oftenest upon the skin close to the lips or the neighboring portions of the face. In other more severe cases the group is larger (Fig. 429), or there are several of them, or the eruption may occur on the cheek, the mucous membrane of the mouth or throat, or, in fact, almost any portion of the cutaneous or mucous surface. (See Herpetic Stomatitis, Vol. I, p. 663.) Preceding the appearance of the vesicles a burning sensation may be experienced. The duration of the attack is a few days or a week, the vesicles tending to rupture and to form crusts which fall off usually without scarring. In very many individuals there is a peculiar disposition to recurrence, generally in the same region, and this persists during years. The diagnosis is readily made from the appearance, the situation, and the tendency to recur. Herpes zoster is distinguished by the location along the course of the nerves, and by the pain.

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[1] Arch. f. Dermat. u. Syph., 1891, XXIII, 633.

[2] Journ. Cutan. and Genito-urin. Dis., 1891, IX, 48; 1892, X, 184.

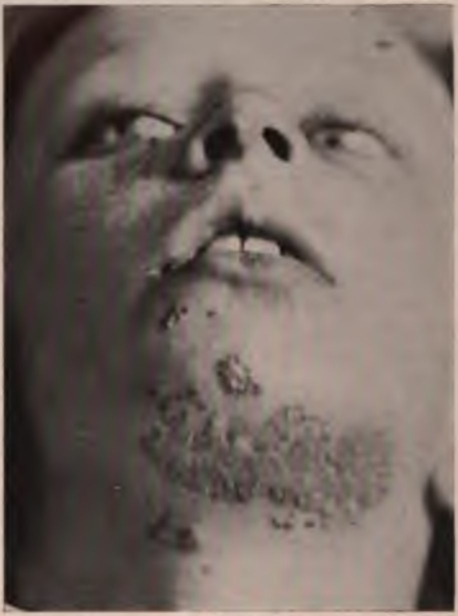

Fig. 429.—Herpes Simplex.

Unusually well-developed eruption effecting chiefly the region of the chin. From a patient in the Children's Hospital of Philadelphia.

In the line of **treatment** it is sometimes possible to abort an attack by painting with flexible collodion, dabbing frequently with camphor water, or applying sulphur ointment. If the vesicles have already developed, zinc ointment, calamine lotion (p. 555), or boric acid and camphor water may be employed.

HERPES ZOSTER

(Shingles)

Etiology.—This is not at all an unusual disease in early life. In its nature it is a nervous one, depending upon an inflammation of a root-ganglion or the sensory nerve connected with it, the eruption being merely the cutaneous manifestation. The causes assigned are very varied, and the disease has been supposed to be a systemic infection. Apparently anything which can produce the inflammation of the ganglion or the nerve may act as the cause.

Symptoms.—These consist in the occurrence of neuralgic pain, accompanied, or followed in a few hours or longer, by the appearance of vesicles scattered along the course of some one nerve or sometimes more than one. The vesicles appear upon one side of the body only; very exceptionally bilaterally. There may be considerable burning and some degree of itching experienced. Very frequently in children the pain is only slight or may be absent. Sometimes moderate fever, malaise or nausea precedes the eruption. The vesicles are split-pea in size, grouped, and situated upon a pinkish-red base. They are for the most part discrete, and their contents become turbid and dry into crusts, perhaps preceded by rupturing. The crusts persist for a week or more and leave temporary staining of the skin. Only occasionally does suppuration occur, the result of irritation by the clothing, or produced in other ways. Although the eruption may involve different parts of the body varying with the case, the usual situation is the trunk, in the course of the intercostal or abdominal nerves; but it is also common on the buttock, thigh, neck and forehead. In the latter situation it may involve the eye. Very often in adults tenderness of the skin and severe neuralgic pain may continue for weeks or months after the eruption has disappeared, but this is an uncommon occurrence in children, and the diagnosis is generally easy. Herpes simplex has a less red base, usually smaller vesicles, and is unattended by sensory symptoms of any moment. The **treatment** in children seldom need be other than local. The affected part should be protected from irritation best by bandaging, after applying a dusting-powder of oxide of zinc and starch. Sometimes the employment of menthol in some form is serviceable.

PEMPHIGUS NEONATORUM

Etiology.—True pemphigus is a very rare disorder, seen after the period of early infancy, and need not receive further consideration here. The condition designated as *pemphigus neonatorum* is certainly closely allied to, if not to be considered a form of, contagious impetigo (*impetigo bullosa*). It is an infectious and somewhat epidemic and contagious disorder occurring in the first 2 weeks of life, and dependent upon some pyogenic microörganism, oftenest the staphylococcus aureus.

Symptoms.—The disease is at first characterized by the appearance of scattered bullæ increasing in size to ½ inch (1.3 cm.) or less in diameter, with a slightly reddened base, and seen oftenest at first upon the hands or face. These bullæ rupture in a few days, or dry and form crusts; but in some cases other crops continue to appear of larger size, and fuse with each other until they are present extensively over the body. The skin of the affected area, from which the epidermis readily strips, is now inflamed and of a raw, red appearance. The general health of the child is little affected at first; but as the disease progresses there is increasing debility and weakness. There may be irregular fever and septic symptoms.

Another variety is that denominated *syphilitic pemphigus*, also a disease of the new born, which is liable to involve especially the palms and soles, although the bullæ may appear elsewhere on the cutaneous surface. It occurs only in weakly wretched infants with other evidences of hereditary syphilis (Vol. I, p. 567).

Course and Prognosis.—The prognosis of pemphigus neonatorum is always uncertain. In the milder cases occurring in infants previously in good health, recovery takes place after a few weeks. The more severe cases may run a rapid course with fever and septic symptoms and terminate fatally in a week or less. The syphilitic cases always die in a few weeks.

Diagnosis.—This usually offers no difficulty. The distinction is to be made between the non-syphilitic and the syphilitic forms by the involvement of the palms and soles in the latter, and the other symptoms of this disease, including enlargement of the liver and spleen. Dermatitis exfoliativa is certainly very closely allied etiologically to pemphigus neonatorum, and the two conditions may occur in the same epidemic, or an infant with one disease may show a change of this into the other. The clinical appearances are, however, different. True pemphigus is to be distinguished from the so-called pemphigus neonatorum by the development of the blebs in a state of considerable size from the beginning, while the eruption of pemphigus neonatorum is small at first and grows gradually from the periphery.

Treatment.—The treatment consists in opening the bullæ, protecting the affected parts, the application of an oxide of zinc and starch powder, or the use of oxide of zinc paste, a weak ammoniated mercury ointment (gr. 10:oz. 1) (0.648:31), or a boric acid ointment. At the time of renewing of the applications the skin should be washed with a 1:5000 solution of corrosive sublimate.

FURUNCULOSIS. MULTIPLE CUTANEOUS ABSCESSES

Etiology.—These two conditions are closely allied and in atypical cases not always clinically to be sharply differentiated from each other. Both depend upon the entrance of pyogenic germs, especially the staphylococcus aureus, into or beneath the skin. Various predisposing causes exist. There is an individual tendency to furuncles seen in many children, otherwise apparently in the best of health; but generally, especially in cases with widespread, numerous small suppurating cutaneous foci, there is a discoverable cause of some sort. There may be evidences of indigestion or of the taking of improper food; or the subjects may be poorly nourished or debilitated by some recent disease, as typhoid or other fever; or there is a local irritation, as from eczema or impetigo. The multiple subcutaneous abscesses of infancy occur chiefly in marantic subjects.

Symptoms.—In the typical furuncle the hair-follicles or sweat-glands are involved; in the abscess the subcutaneous tissue. The former may occur at any age; be small or large; single or very numerous. There results a painful induration which suppurates and has a central slough. Multiple subcutaneous abscesses are peculiar to infancy; numerous, widespread, large or quite small. These abscesses, at first perhaps hard, soon become soft and suppurating, but without the acuminated character of the boil and there is no central slough. Intermediate between these two are, however, the very numerous cases seen principally in infancy, less often later, with widespread small suppurating foci, which have no special resemblance to a boil, and yet do not occur in the subcutaneous tissue, but rather consist of small abscesses or ulcerations in the substance of the skin itself. Considerable constitutional disturbance with fever attends the production of furuncles, as well as of the large subcutaneous abscesses; while the small numerous abscesses of the skin itself generally exhibit but little fever, and the constitutional symptoms are those of the primary disease.

The **prognosis** of these purulent processes is generally good, although it not infrequently happens in the case of very widespread and constantly recurring suppurating foci in debilitated infants that great exhaustion and even death follow.

Treatment.—The treatment of any of the forms consists in incision and discharge of the pus as early as possible. Before the condition indicating the presence of pus has been reached, an effort should be made to promote absorption by the application of ichthyol ointment (10 to 15 per cent.). If suppuration seems certain, the softening of the skin may be hastened by hot compresses or antiseptic poultices. When there is a tendency to the appearance of repeated crops of suppurating areas, tonic remedies, such as iron, cod-liver oil, or arsenic, are strongly indicated, combined with regulation of the diet and the removal in general of any possible cause. Among substances recommended for a supposed specific action are yeast and calcium sulphide. It seems questionable whether these possess any real value. In addition the neighboring skin should be kept as aseptic as possible by the washing of the affected region daily with a bichloride of mercury solution (1 : 5000) to prevent autoinoculation. For severe recurring cases vaccine treatment with autogenous vaccines should be tried.

IMPETIGO CONTAGIOSA

Etiology.—The disease is an infectious one, both contagious and autoinoculable, and depends upon the action of some of the pyogenic bacteria, oftenest the staphylococcus aureus or a streptococcus. It would appear from the studies of Sabouraud[1] that there are at least two forms. The most frequent and most contagious is that described by Tilbury Fox.[2] This is dependent upon a streptococcus, and is characterized by lesions which are principally vesicular or bullous at the onset. The second form is that of Bockhart.[3] It is less frequent and less contagious than the other, is dependent upon the staphylococcus and is pustular from the start. Impetigo contagiosa occasionally occurs in adults, but is most frequent in infants and young children. Generally several children in a family are attacked; or a considerable number when the outbreak is in an institution for children. Although entirely healthy subjects may suffer, those with impaired general health are most likely to become affected.

Symptoms.—In the typical form of the disease as described by Fox, and as discussed here, the lesions usually start as small vesicles or vesicopapules, but soon increase in size, perhaps reaching ½ inch (1.3 cm.) in diameter. They are not fully distended, and their contents become a yellowish sero-purulent fluid, and later dry into yellowish crusts in from 2 to 5 days (Fig. 430). The crusts are commonly described as seeming to be "stuck on" in the centre, with the edge slightly raised. The eruption as a rule develops especially on the face, scalp and hands; but may spread by autoinoculation to other regions, particularly the legs, feet and forearms. The lesions may be quite numerous or few only, and are generally discrete, although some degree of coalescing may occur when they are very much crowded. Under the crusts the surface is moist and red, but becomes dry after these have fallen. Very slight evidence of inflammation is seen in the skin surrounding the lesions. There is little itching, and no constitutional symptoms are present. The glands in the neighborhood of the lesions may become inflamed.

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[1] Annales de dermat. et de syphilogr., 1900, 4 s., I, 62; 320.

[2] Brit. Med. Journ., 1864, I, 467.

[3] Monatsh. f. prakt. Derm., 1887, VI, 450.

From this description of the more typical cases there may be variations seen. Sometimes in debilitated children the deeper portions of skin become involved and ulceration occurs. In other cases, by confluence of several lesions, there may develop a raw area healing in the centre, but with a circinate and extending border (*impetigo circinata*).

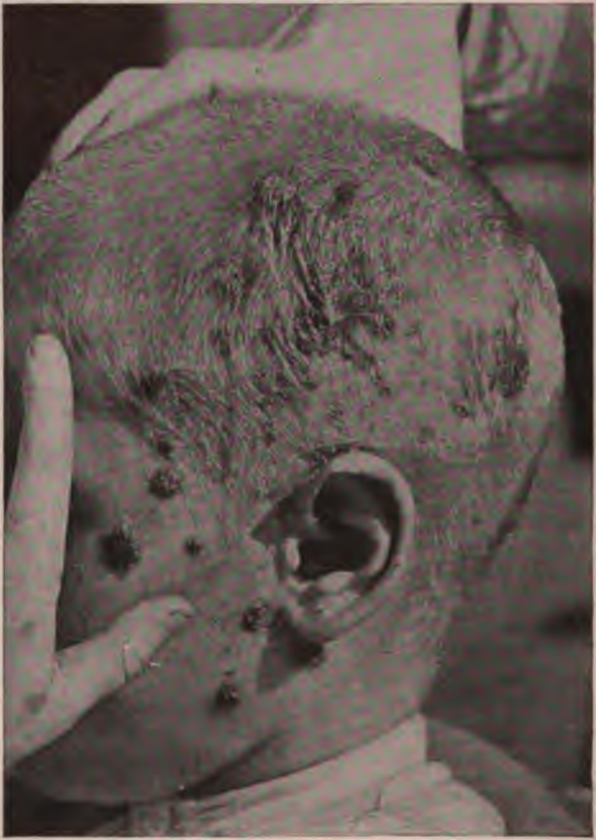

FIG. 430.—IMPETIGO CONTAGIOSA.
(*Hartzell, Diseases of the Skin*, 1917, 192.)

There is also a bullous form seen (*impetigo bullosa*) in which the lesions are prone to remain bullous especially on the extremities and the genitals; and in some cases only bullæ may be found, sometimes as large as an egg, without any tendency to the formation of crusts. The mucous membranes are sometimes attacked in this variety. This form of the disease is seen oftenest in young children and in infants; and [the pemphigus neonatorum (p. 565) described as attacking the latter is probably a form of impetigo.

Course and Prognosis.—In the ordinary form this is entirely favorable under treatment, and recovery takes place in 2 or 3 weeks, but the course may be much prolonged by repeated autoinoculation.

The bullous form is often very tedious and may terminate fatally. In cachectic subjects the lesions of impetigo may assume an ecthymatous character, the inflammation extending deeply, the skin being much infiltrated, an extensive red area surrounding the lesion, and a large dark crust forming which covers deep ulceration.

Diagnosis.—The diagnosis of typical cases of the ordinary form of impetigo is generally easy. It rests upon the occurrence in several children of the house; the position of the lesions; the disposition to spread by autoinoculation; the absence of much itching; and the formation of thick yellowish "stuck on" crusts. Eczema could be mistaken for it, but exhibits much smaller vesicles and crusts, severe itching, and distinct evidences of infiltration and edema of the skin. The bullous form of impetigo may strongly suggest varicella. It differs in the longer course, the greater size of the lesions; and the absence of the areola. As seen in the so-called pemphigus neonatorum it has already been discussed.

Treatment.—This consists in opening the vesicles and bullæ, removing the crusts by warm water and soap, and the application of an antiseptic ointment, one of the best of which is of ammoniated mercury (gr. 15 to 20) (1 to 1.3) : oz. 1 (31). This should be kept constantly applied. The frequent employment of a lotion of corrosive sublimate (1 : 5000) or washing with a superfatted sulphur soap is also of benefit in preventing further spread of the disease. Frequent changing of the underclothing is of importance. In the bullous form, if extensive, a lotion of boric acid may be used and a boric acid ointment applied.

NEVUS

(Angioma; Birth Mark; Mole)

Classification and Symptoms.—Under the name of "nevus" are included several varieties of congenital growths of the skin, either vascular, pigmented, or of other nature. Of these the most important is the *vascular nevus*. It is either seen at birth, or develops soon after, and may vary greatly in size and character. It may consist of very small reddish or bluish spots; flat or slightly elevated; pin-head size or a little larger; disappearing on pressure, and composed of a group of small dilated capillaries (*telangiectasis*). A larger area of similar nature constitutes the well-known "port-wine stain" (*nevus flamineus*). This may be of varied size and color, and occur on any part of the body, although the temples and cheeks are favorite sites. In another form of nevus (*angioma cavernosum*) the arterioles are decidedly dilated, there is overgrowth of the surrounding connective tissue, the affected area is elevated above the skin about it, and the color is purplish.

The *pigmented nevus*, or *mole*, is oftenest situated on the face, neck, or back, and may be single or multiple, of varying size, and flat or hypertrophic. Not uncommonly it is more or less covered by coarse hair. The mole is composed of a great overgrowth of cuboidal epithelial cells (nevus cells) in the cutis, together with an unusual deposit of pigment in the rete and corium.

Course and Prognosis.—The prognosis of nevi is variable. Some of the smaller may disappear as the child grows older. It is a possibility for them, under the influence of irritation, to develop eczema or ulceration; while in other cases they may exhibit rapid extension in size and undergo a malignant transformation. This is particularly true of the thickened nevi of a cavernous nature. Moles have little tendency to change, but the hypertrophic form may develop carcinomatous changes. Port wine stains offer little hope of improvement.

Treatment.—This belongs rather in the province of the surgeon or the dermatologist. The small vascular nevi and the moles may well be let alone unless in a position where they are a source of disfigurement. Treatment of nevus is of the nature which will produce the closing of the vessels in the vascular growths, or the removal of the pigment in the others. Electrolysis has been used for this purpose, or more recently the application of carbonic dioxide snow. Various caustics have also been used, as has exposure to the *x*-ray. If rapid growth of the vascular nevus or mole begins, surgical removal is to be recommended.

LENTIGO

(Freckles)

This so common condition, consisting of small numerous deposits of pigment in the skin, is a matter of no serious importance, but often a cause of mortification to children and especially to their parents. Freckles are more frequent in blondes, are usually not seen before the age of 3 years, and tend to disappear as the patient grows older, although this is by no means always the case. They develop especially in summer, and on the portions of the body exposed to the sunshine (p. 577, Fig. 433). Prevention should be attempted, when the matter is of sufficient moment, by the employment of a veil and a broad-brimmed hat. For their removal an application may be made of a lotion of compound tincture of benzoin and glycerin each fl.dr. ½ (1.8) and aq. rosæ fl.oz. 3 (89); or one of sodium biborat. dr. 1 (3.9) acid acet. dil. fl.oz. ½ (14.8), aq. rosæ fl.oz. ½ (14.8). Stronger lotions may be used containing bichloride of mercury in water or diluted alcohol (gr. 6 (0.39): fl.oz. 4 (118), but with caution lest the action be too vigorous.

VERRUCÆ

(Warts)

Classification and Symptoms.—Verruca may appear in two forms: the ordinary wart (*verruca vulgaris*), and the plane wart (*verruca plana juvenilis*). They are alike in structure, exhibiting hypertrophy of the papillæ with an overgrowth of connective tissue. The process starts in the rete. They appear to be infectious and autoinoculable, and probably depend upon some unknown microörganism. Beyond these facts the cause of their occurrence is unknown. The period of incubation is probably several months.

Verruca vulgaris appears in the form of a hard elevation of varying size and height; sometimes flat; sometimes filiform, or partially broken up into a number of small lobules. There may be one or numerous warts. They occur especially in children and oftenest upon the hands, scalp and face. Frequently a single wart is first to appear, and then numerous others in the vicinity. They produce no disturbance except that they may become inflamed by mechanical irritation. Their course is very uncertain. Sometimes they appear to grow rapidly, sometimes only very slowly, and they may last for months or even years, and may then disappear without treatment.

Verruca plana juvenilis consists of a pin-head sized or sometimes larger, flat, slightly elevated, and round or irregularly shaped growth of a yellowish or pale-brownish color. The warts are generally very numerous and sometimes confluent. They occur chiefly on the face, hands or forearms. Their surface is more delicate than that of the common wart, and easily injured by scratching. They are frequently found associated with the ordinary wart.

Treatment.—The treatment frequently presents considerable difficulty. Internally arsenic is often of value for both forms of warts, but especially in the juvenile form. Locally verruca vulgaris may be treated in various ways. The wart may be touched daily with strong nitric acid, glacial acetic acid, or trichloracetic acid, taking care that the fluid does not spread to the surrounding skin. Freezing with carbonic dioxide snow or exposure to the *x*-ray is also of service. A less energetic treatment, suitable also for the plane wart, is an ointment of salicylic acid (2 to 4 per cent.).

ICHTHYOSIS

Etiology and Pathology.—This is probably nearly always a congenital disorder, although it usually does not become apparent until about the end of the 1st or 2d year of life. Cases showing the symptoms severely at birth are rare. A hereditary and familial tendency is often very evident. In its anatomical nature the condition is rather a malformation of the skin than a disease. There is a thickeneng of the epidermis due to an increased formation of squamous epithelial cells, and the sweat glands and sebaceous glands are diminished in number.

Symptoms.—In cases of average severity these consist in the widespread covering of the body with grey, yellowish, or yellowish-brown scales, loose at the edges, and suggesting the scaly covering of a fish. The localities most markedly affected are the extensor surfaces of the extremities, especially the lower, and the trunk. The flexor surfaces and the face are involved to a much less degree. The skin is harsh and dry and somewhat scaly and the hair is dull and dry. The palms and soles are smooth, but exhibit deep furrows. There is no itching or other subjective symptoms. The disease is always better during the summer season.

By no means all cases answer exactly to this description. In some the disorder is much milder and exhibits little more than an unusual roughness of the skin chiefly on the back and extremities, with prominence of the follicles, and with the scaliness not exceeding a slight branny desquamation; all these conditions being most noticeable in the winter season, at which time eczema is prone to develop as a complication. On the other hand, in the more severe cases, the scales persist in the form of thick plates with deep furrows in the skin separating them, and often with fissures near the joints; the flexor surfaces are involved, and the scalp and face are rough, dry and scaly. In some of these cases the appearance of the patches with the furrows between them gives rise to the appellation "alligator skin."

Rarely the new-born infant may exhibit ichthyosis in a very advanced stage of development (*congenital ichthyosis*). In this, the most severe form of the disease, the whole surface of the body is found covered at birth with thick, plate-like masses of scales and exhibits numerous furrows and fissures. The skin is dry and thickened and the normal folds largely obliterated. The ears and nose may be nearly closed by the epithelial scales; ectropia is present; nursing is difficult or impossible on account of the interference with opening the mouth, and death usually takes place from inanition in a few days or a week. A less severe degree of this congenital or fetal ichthyosis may be observed in which life continues for a longer time.

The **course** of ichthyosis is chronic, and the disease incurable, although often capable of being much benefited by treatment and improving also in warm weather when the glands of the skin are more active. The so-called congenital form is soon fatal except in the mildest cases. The **diagnosis** is always easy, there being no other malady resembling it. I have, however, known an unwary and over-zealous health-officer order an ichthyotic child convalescing from scarlet fever to be kept in the room on account of the continued "dangerous" scaling.

Treatment.—Measures must be employed to keep the skin as soft as possible. Warm baths should be taken daily. The addition of bran, borax, or hyposulphite of soda to the water is sometimes of service. The bath should be followed by inunction with any bland oily substance. Internally thyroid extract has been found of temporary benefit. Cod-liver oil, arsenic, or other tonic remedies should be given if the health is below normal.

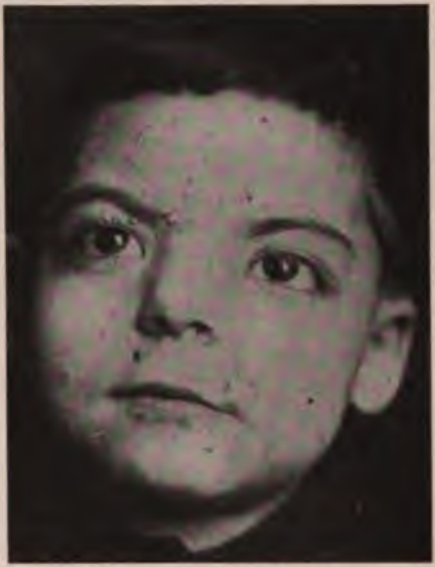

Fig. 431.—Molluscum Contagiosum.

From a patient in the Children's Hospital of Philadelphia. Scattered lesions are seen chiefly on the eyelids and about the mouth.

MOLLUSCUM CONTAGIOSUM

This is a distinctly infectious disorder seen at all ages, but much oftenest attacking children, and sometimes occurring endemically in schools and institutions. The nature of the germ is not known, but the disease has been transmitted by experimental inoculation. The lesion consists of small masses of proliferated epithelial cells, divided into lobules separated by connective-tissue septa. The upper central part of the growth is composed of white, opaque degenerated epithelial cells which can be pressed in a single mass from the opening at the summit. This constitutes the so-called "molluscum body."

Symptoms.—Clinically the lesion appears as a small, shining, yellowish or whitish nodule of pin-head size up to that of a pea, with a minute opening at the slightly depressed centre (Fig. 431). The nodules are globular or flattened elevations, but sometimes pedunculated and larger. There is no evidence of inflammation, unless the growth has been irritated, when suppuration may occur. As a rule there are no subjective symptoms, but in some cases there may be itching. The nodules may disappear by shrinking, or may discharge the central mass and dry up; although more frequently they run a chronic course and last an indefinite period. They are situated oftenest on the eyelids, forehead, neck, and genitals, but may occur elsewhere. Generally only about a dozen growths are present at one time, but new ones continue to develop as the old ones disappear. The **diagnosis** is generally easy. It is to be made especially from warts; molluscum contagiosum differing from these bodies in the normal skin which covers the growth, and in the depressed centre with punctiform central opening.

Treatment.—This is generally easy. If the lesions are few in number, each one may be treated by incision followed by expression of the central mass, and then a cauterization of the cavity; or trichloracetic acid may be carefully applied, or the electric needle employed. If the growths are numerous, an ointment of sulphur, or of ammoniated mercury (4 per cent.), may be rubbed in vigorously once or twice a day, or a 0.8 per cent. dilution of liquid formaldehyde applied.

TUBERCULOSIS OF THE SKIN

The subject is of such importance that justice can be done it only in works upon dermatology, and but brief mention will consequently be made of it here. Some of the various manifestations are rare and reference to them may be omitted; others are common at any period, and some oftenest seen in early life. The lesions are divided into those which are produced directly by the tubercle bacilli; and others, the *tuberculides*, which would appear to be the result of the action of the toxin of tuberculosis present in the body, the bacilli themselves not being demonstrable in the lesions. The histology is the same in all the varieties, and is that of the ordinary tuberculous nodule; and it is maintained by many that all of them are in reality the product of the local action of the germs.

1. Lupus Vulgaris. **Etiology.**—This disease often begins in childhood; is seen most frequently among the poor; and is not often observed in this country, although common in Europe. A debilitated condition of health predisposes to it, as does the occurrence of some one of the infectious diseases. In a large proportion of cases tuberculosis existed in the parents, or had been present elsewhere in the body of the patient.

Pathology.—The germ reaches the skin through one of several ways; either by direct local infection from a tuberculous source in the patient's body, or outside of it; or by the blood current. Which is the most frequent method is still a subject of discussion. The histological process consists in the deposition around a blood-vessel of granulation tissue with giant cells; and with tubercle bacilli present in very scanty numbers, or discovered only by inoculation. The nodules are found in the papillary layer or deeper in the skin.

Symptoms.—Clinically the lesion appears as a brownish-red, flattened, slightly elevated, indurated area. Examination shows that this is composed of small confluent nodules of pin-head size, or slightly larger.

These constitute the characteristic feature of the disease. The brownish-red color and the nodular character are well revealed if a flat piece of glass is pressed upon the patch. As time passes the surface of the diseased region may become ulcerated and crusted, or may exhibit a development of cicatricial tissue, or the process may result in a warty growth oftenest situated upon the extremities. The patches are most commonly single, but may be several in number, and especially after acute infectious fevers, may be numerous over the body. The disease appears most frequently on the face, particularly on the end of the nose or on the cheeks; but any part of the cutaneous surface may be involved. The mucous membrane, too, principally of the nose and mouth, may be attacked primarily or secondarily.

Course and Prognosis.—The course is very slow, perhaps years being required to produce a patch of much size. There are periods, too, in which the growth is quiescent. The lesion extends gradually from the periphery by the production there of fresh nodules, and if neglected may cause extensive destruction of tissue, with formation of disfiguring scars. This scar-tissue is the result of the effort of the lesion to heal spontaneously; but fresh infiltration is likely to appear in it. The prognosis on the whole is unfavorable, improvement taking place only after long-continued and persistent treatment. It is best in children because of the lesser degree of advancement of the case when coming under observation. There is always the possibility of tuberculosis developing elsewhere in the body, if not already present.

Diagnosis.—The recognition of the disease rests upon the long continuance of the lesion and the discovery of the characteristic lupus-nodules. The latter serve to distinguish lupus from some forms of cutaneous syphilis.

Treatment.—This is a matter best placed in the hands of a dermatologist. Various remedies have been employed, including the use of the *x*-ray; Finsen light; excision; cauterization, and the injection of tuberculin. The choice of these depends largely upon the character and position of the lesion. Probably, when possible, early excision is to be preferred.

2. Scrofuloderma. **Etiology.**—By this title may be designated the lesions of the skin, of an actual tuberculous nature, which depend upon the presence of tuberculosis in an adjacent lymphatic gland or portion of osseous tissue, the skin being directly infected from these foci; or which less often develop independently of any such focus. The lesions may occur at any age, but oftenest in early life, and even in early infancy. They consist of localized tuberculous infiltrations, with numerous giant cells and very few discoverable tubercle bacilli.

Symptoms.—When connected with tuberculous bone or lymphatic glands, the lesions appear as hard, pointed swellings of the skin of a purplish-red color; or they may develop as subcutaneous tuberculous infiltrations occurring independently of any underlying primary lesion. The mass soon softens, flattens, and opens by one or several small ulcers covered with pale, semi-translucent, soft granulations. When more than one they are connected by sinuses with each other as well as with any underlying tuberculous focus present. The area grows in extent by the enlargement of the ulcer, or by the formation of new ones, with overhanging edges and with purple-red, thin, and often undermined skin between them. The lesions are found oftenest in the neck and groins, or upon other positions where caseous glands or necrotic bone exists. In infants they are seen oftenest on the face.

The **course** of the disease is very chronic. There is little tendency to healing shown unless the cause is removed, any scar-tissue which forms being very likely to break down again after a short time.

The **diagnosis** is usually easy, based upon the symptoms described, together with the presence of neighboring tuberculous foci, and the association of such conditions as blepharitis, keratitis, purulent otitis and rhinitis, and the like.

Treatment.—This consists primarily in removing any underlying osseous or glandular disease present. The employment of the *x*-ray is often of benefit to the cutaneous lesion, as is also the application of iodoform or boric acid preparations; and treatment with tuberculin has been employed. Of greatest importance is the improvement of the general health by suitable climatic, dietetic, and hygienic measures, and by the administration of cod-liver oil.

3. Lichen Scrofulosorum.—The title designates a form of cutaneous tuberculous disease seen especially in children, but rare in this country. It is one of those generally called a "tuberculide," without evidence of tubercle bacilli in the lesion, although the presence of these has been claimed by a number of investigators. It sometimes follows an attack of one of the exanthemata. Frequently other forms of tuberculosis are readily discoverable; and even when not, the presence of lichen scrofulosorum is an indication that tuberculosis probably exists elsewhere in the body. The condition is believed to be produced by the toxin of tuberculosis.

Symptoms.—The disease is characterized by the appearance of pinhead sized nodules of a yellowish-brown or reddish color, firm, but little elevated, and occurring isolated or oftener in patches up to 1 to 2 inches (2.5 to 5.1 cm.) in diameter. There may be a great many or a limited number of such groups. The basis of each nodule is a hair-follicle; the summit may be slightly scaly. The nodules are seen on the trunk, and are unproductive of subjective symptoms.

The **course** is very chronic. It may continue for months without change and may last for years. The original patches gradually disappear, usually without trace, but new ones develop. The prognosis is good in cases under treatment. The **diagnosis** rests upon the occurrence on the trunk, the absence of itching, the age of the patient, and often the evidence of tuberculosis elsewhere in the body. **Treatment** consists in improving the general health especially by cod-liver oil, and in the application of an ointment containing thymol or subacetate of lead.

4. Acne Scrofulosorum (*Papulo-necrotic tuberculide; Acne necrotica*). —This is an affection not common in this country, allied to the lichen scrofulosorum described and often associated with it. It is uncertain whether the condition is a toxic-tuberculide or produced directly by the tubercle bacillus. It has sometimes followed one of the infectious fevers. The **lesions,** which continue to appear in crops, differ from those of lichen in that they occur on the face and extremities, instead of the trunk; are larger in size; firm, hard, papular, bluish-red, and crusted at the summit, due to the development there of a small ulcer. The process consists in an inflammation of the hair-follicles. The circulation in the extremities is poor and the hands and feet blue and cold. Other tuberculous manifestations are nearly always present. The **course** of the disease is very chronic, and as the lesions heal small scars remain. Under treatment the eruption disappears, but is liable to return especially in the winter season. The **treatment** consists in the ordinary methods employed for the improvement of the general health in tuberculosis and the use of some antiseptic application.

RING-WORM

(Tinea)

Etiology.—The parasitic disorder bearing the name of ring-worm was once supposed to depend upon a single species of vegetable microörganism. It has, however, been shown by Sabouraud[1] that there are more than 40 species of fungus belonging to the same family which are capable of producing the disease in some form. For practical purposes in this connection but 2 genera need be mentioned: the *microsporon Audouini*, or other species of this genus, a small-spored fungus; and the *tricophyton*, a large-spored fungus. The latter is further divided into *endothrix* and *ectothrix*, according as the fungus is found within the shaft of the hair, or on its surface. The species of the parasite varies with the locality. On the scalp the microsporon is the cause in the large majority of instances, perhaps 90 per cent.; while elsewhere on the body the tricophyton is nearly always the active agent. The age of the patient has a predisposing influence in determining the locality; ring-worm of the scalp being largely limited to children, especially between the age of 5 and 10 years, and seen rarely after puberty; while ring-worm of the body can occur at any age, although oftenest in children.

Ring-worm is distinctly contagious from one patient to another, either directly or through clothing, articles of the toilet, and the like, its primary source being not infrequently some of the domestic animals. The microsporon Audouini is a human species, but occasionally other species of this genus may be acquired from the dog, cat, or horse, producing ring-worm of the scalp and occasionally of other parts. The tricophyton, the most frequent cause of ring-worm of the body, is very commonly acquired from other animals, as the cat, horse or dog.

Pathological Anatomy.—The growth of the fungus starts in the epithelium and results in a patch of greyish scales; and later, if the scalp is the seat of the lesion, spreads to the hair-follicles and the hair. Microscopic examination of the hair after previous treatment with liquor potassæ reveals the characteristic spores and mycelial threads, the number and size of these depending upon the species.

Symptoms.—Clinically the disease as seen in children is divided into (1) Tinea tonsurans, or ring-worm of the scalp and (2) Tinea circinata, or ring-worm of the general surface.

1. Tinea tonsurans.—In this extremely common affection there are found one or more rounded grey scaly spots from a fraction of an inch up to 2 inches (5.1 cm.) or more in diameter, which are partially denuded of hair and exhibit a number of broken-off, opaque, brittle, thickened, hair-stumps (Fig. 432). These differ from healthy hairs which have been cut, in their lack of elasticity and their softness when felt with the finger, and in that they point irregularly in different directions. They can be very readily pulled out with forceps. Sometimes the hairs have been broken off close to the skin, producing the "black-dot" variety of ring-worm. The degree varies to which scaling occurs. It may be so abundant that the stumps of the hair are largely concealed, or so little that it is scarcely apparent. Occasionally in some of the latter cases no hair-

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[1] Les Teignes, 1910.

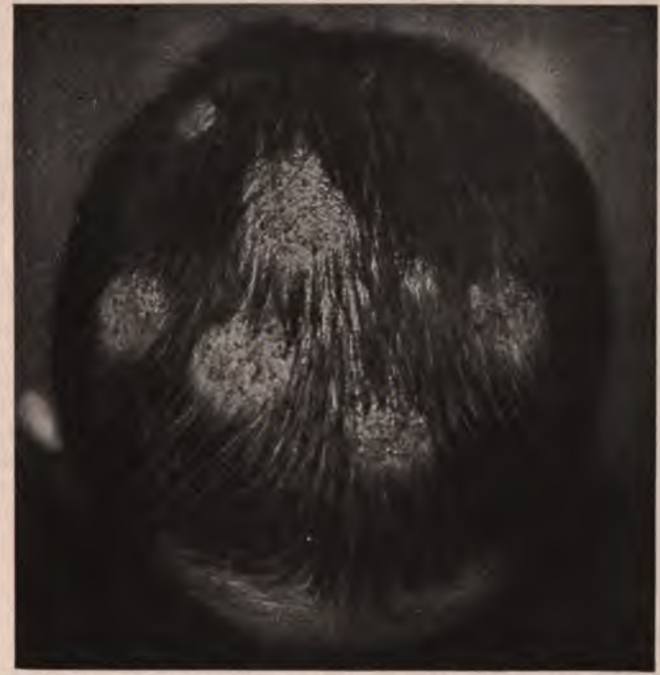

FIG. 432.—RINGWORM (TINEA TONSURANS) OF SOMEWHAT INFLAMMATORY TYPE. (*Stelwagon, Diseases of the Skin, 8th Ed.*, 1171.)

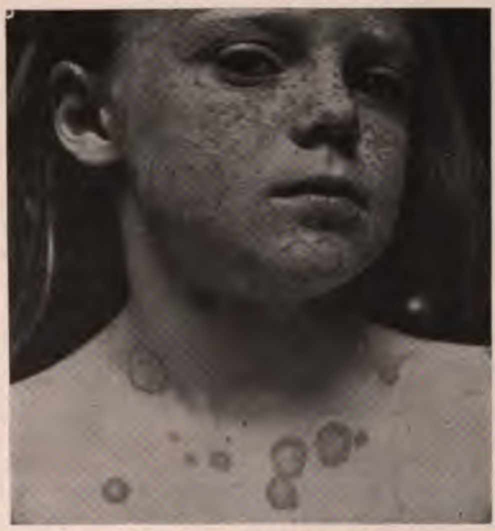

FIG. 433.—RINGWORM OF THE BODY.

Showing the rather uncommon occurrence of a double ring. Illustration also shows a marked example of freckles. (*Stelwagon, Diseases of the Skin, 8th Ed.*, 1163.)

stumps at all may be detected except around the edges, and the condition may suggest alopecia areata (*tinea decalvans*). Sometimes inflammation is set up in the patch, producing deep-seated, boggy, suppurating lesions (*tinea kerion*) which perhaps break down and result in abscess.

2. Tinea Circinata.—The lesions here are situated oftenest upon the face, neck, hands and forearms, rarely on the palms and soles, and may occur with or without involvement of the scalp. The lesions appear as scaly, pink, slightly elevated and distinctly circumscribed small spots. These enlarge into ring-shaped areas by extension of the periphery, this being a trifle elevated and reddened in color, while the centre is paler or whitish and very slightly scaly, the result of the healing process going on there (Fig. 433). There are usually only 1 or 2 patches, but the number may reach 10 or more. They vary in size from ½ inch (1.3 cm.) up to several inches in diameter. When numerous and close together some confluence may occur; but generally they are discrete. Considerable itching may at times be present. Variations may be seen from this description; in some cases the periphery being more inflammatory, with papules and vesicles; rarely a double ring being observed; sometimes the centre showing little tendency to healing; or the whole area being inflammatory. Decided inflammation, as in the tinea kerion of the scalp, is of uncommon occurrence. The tricophyton sometimes but not often attacks the nails; which grow opaque, rough, thick and brittle.

Course and Prognosis.—The extension of *tinea tonsurans* is generally rather slow, several weeks or months being required before the patches reach their full development. After this little change may take place, or new spots may appear; or confluence may occur between the affected areas until much of the scalp is involved. The ultimate prognosis is good, but the time when recovery will be accomplished, even under treatment, is most uncertain. Untreated cases may last for years. The disease finally disappears, and the hair grows again as the patient reaches puberty. When the eruption is limited to a few areas and treatment is energetically and faithfully carried out, recovery may take place in a few months, but often a year may be required. The disease is most amenable to treatment when dependent upon the ectothrix variety of the tricophyton. The occurrence of kerion is often curative.

Tinea circinata likewise develops rather slowly and the patch, after reaching its maximum size, tends to remain stationary for a time and then to disappear. Treatment is usually efficacious after a few days or weeks. Recovery from both forms of tinea is usually without any permanent trace of injury remaining.

Diagnosis.—The recognition of tinea circinata is generally easy, based upon the circinate character with the pale healing centre. A microscopic examination will settle all doubtful cases. The diagnosis of tinea tonsurans is always easy in typical cases, the combination of scaliness, loss of hair, and broken hair-stumps being characteristic. At times, however, confusion can arise, especially with alopecia areata and favus. The former never exhibits any broken stumps, and the skin is smooth and shining. Favus does show broken hair-stumps and baldness, but is recognized by the sulphur-yellow, cup-shaped crusts. The scaling and loss of hair in seborrhea is diffuse and without broken hairs. Psoriasis produces rounded patches, but they are more widely scattered and without the characteristics of tinea. The inflammatory type of tinea tonsurans may suggest inflammations of other nature, especially eczema, but there is not in these latter the localized loss of hair with little stumps remaining.

Treatment.—Tinea circinata is usually easily cured. Any scaliness should be removed with soap and water and the ring-worm treated with a parasiticide. One of the best is tincture of iodine, ½ strength for children, painted on the spots daily for 3 or 4 days. If the disease is on a situation where the iodine would be disfiguring, a lotion of bichloride of mercury (0.5 per cent.) may be dabbed on the spots several times daily, or an ointment applied consisting of precipitated sulphur dr. ½ (1.9) with Lassar's paste (p. 545) oz. 1 (31).

Treatment for tinea tonsurans is well-known to be exceedingly troublesome and discouraging, although generally successful if persisted in. The slowness in obtaining a cure depends upon the difficulty in making the remedies penetrate deeply into the hair follicles. The hair should be cut close or shaved over the entire head and kept so; or, if this cannot be done, then certainly over and in the neighborhood of the patch, although the spread of the disease is more difficult to control if the latter plan is adopted. The scalp should be washed with soap and water, an antiseptic soap being often preferable, and all broken or loose hairs be pulled out with forceps, and after this the parasiticide should be applied and well rubbed in for 5 or 10 minutes. This should be repeated once or twice daily. To prevent the spread of the infection to other children, the patient should wear, covering the scalp, a close-fitting cap of some material which can be boiled; and the greatest care should be taken that all the soap, towels, hair-brushes and other toilet articles are individual ones. The remedies which have been prescribed are very numerous. Among those most frequently recommended may be mentioned ointment of sulphur (gr. 30 (1.9) or more : oz. 1 (31)); tincture of iodine; ointment of iodine (dr. ½ to 1 (1.8 to 3.9) : oz. 1 (31)); ointments with oil of cade or other tarry substance, or of ammoniated mercury, salicylic acid, or chrysarobin. The last must be used carefully to avoid undue irritation. A moderate degree of this is, however, beneficial, as it sets up an inflammation in the interior part of the follicles, and the diseased hair drops out. The strength of the preparation used should vary with the age of the child, the duration and extent of the disease, and the amount of reaction which is produced.

Probably by all odds the most efficacious treatment is the employment of the *x*-ray, as first recommended by Sabouraud and Noiré.[1] This method must, of course, be used by one expert in its application. By means of it depilation is produced readily, the fungus coming out with the hair, and complete cure may be obtained in from 3 to 4 months. Only one such treatment is generally needed, unless the disease is widespread. While the hair is falling after the treatment, an ointment containing a mild parasiticide, such as sulphur or ammoniated mercury, may be applied to the scalp daily, in order to prevent the redevelopment of the lesion from any infected hairs which may have chanced to remain.

FAVUS

(Tinea favosa)

Etiology.—This is a rare affection in this country, seen chiefly in immigrants from Russia, Poland, or any other lands where it is very common. Lack of cleanliness is a prominent predisposing cause. It may appear at any age, but it is uncommon for it to begin on the scalp after puberty. It is dependent upon a mould-like fungus, the Achorion

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[1] Presse méd., 1904, XI, 825.

Schönleinii, and is decidedly contagious, although less so than ring-worm. It may be acquired from other persons, or from certain animals, among them dogs, cats, mice, rabbits, fowls, and less often horses and cattle.

Pathological Anatomy.—The fungus consists of mycelium and spores which first develop in the upper part of the hair-follicles, and spread to the neighboring epidermal portion of the skin. After a time it breaks through the upper layer of the epidermis and produces a cup-shaped cavity filled with a sulphur-yellow mass. The hair shaft is invaded likewise, although not to the degree seen in tinea tonsurans. The hair falls out and often the follicles atrophy and the growth of new hair is permanently prevented. The parasitic nature of the disorder can be readily discovered by the microscope, a portion of the crust having been previously soaked in liquor potassæ for a few minutes.

Symptoms.—The disease attacks usually the scalp. Here it first appears as one or more small yellowish points which soon enlarge and become sulphur-yellow cups, from the depressed centre of each of which a hair projects. The spot grows slowly, reaching the size of a small pea. Gradually in cases of long duration the cups become more or less confluent into yellowish crusts of an inch (2.5 cm.) or more in diameter, and new points of infection appear until in severe cases much of the scalp may be involved. When the crusting is extensive, a peculiar mousey or musty odor may be produced. Beneath the yellowish deposits, if lifted off from the head, there is found a slight inflammation with some serous exudation. Suppurating points sometimes develop near the periphery, or perhaps over the whole area of the patch. The hairs over the seat of the disease are dull and fall out, split, or break off readily. In the region where the affection has existed longest, portions of the yellowish deposit may finally fall off, leaving a nearly hairless area with numerous scattered yellowish points upon it.

Upon the body favus is much less common, and is generally a sequel to the involvement of the scalp. Although sometimes appearing in circinate areas, it usually forms thickened, rough, crusted patches as on the scalp. Rarely the nails are attacked and become thickened and irregularly split, and in their substance can be seen yellowish circumscribed spots. Favus is practically without subjective symptoms, except a varying degree of itching, which occasionally is troublesome.

The **course** of the disease on the scalp is very slow, months often being required before any considerable spread of the affection is developed. On other areas of the body the extension of the disease is often rather rapid. The **prognosis** for the body-surface is good under treatment, and sometimes even without it; but for the scalp it is unfavorable. Here the disease is extremely resistant to treatment, and in advanced cases is practically incurable. Even the patient recovering may have permanent loss of hair in the affected region. The **diagnosis** is easy, there being no other affection which produces the sulphur-yellow cups with loss of hair. The **treatment** is the same as for ring-worm.

TINEA VERSICOLOR

(Pityriasis Versicolor)

Tinea versicolor depends upon the presence of the microsporon furfur. It is not often seen in children, and not until after the age of 7 or 8 years. It is only exceptionally contagious. The occurrence of free perspiration is an important predisposing cause. The fungus consists of mycelium and spores which grow in the horny layers of the epithelium.

Symptoms.—The disease appears as faint yellowish or pale, small, brownish spots, little if at all elevated, slightly scaly, and fusing into much larger areas, and situated chiefly upon the thorax and arms. The hair-follicles are not involved. Very slight itching may be present. The discoloration is readily removed by scraping, leaving no redness of the skin beneath. The course is very slow, months being required before patches of any considerable size appear. The prognosis is good, the disease being generally easily cured, although relapses occur readily. The diagnosis is easy, the recognition of pigmented spots of other nature being made by the absence of the fungus on microscopical examination. **Treatment** consists in the employment of an ointment of precipitated sulphur (1:8), antiseptic soap, or a lotion of hypophosphite of soda (1:32). The remedy should be applied at least twice a day. Relapse is to be guarded against by frequent changing and very thorough disinfection of the underclothing.

SCABIES

(Itch)

Etiology and Pathology.—Scabies is the result of the infection of the skin by an animal parasite attacking only the human race, the acarus scabiei or sarcoptes scabiei. There is a similar affection of domestic animals which may rarely be communicated to man, but differs in some respects in the symptoms produced. Scabies appears at any age, and is very contagious, but requires close contact with an infected person, or with the bed-clothing or body-clothing. Hence, it is acquired oftenest by those who have slept with persons suffering from the disease, or in beds that have been occupied by them. The female acarus, which is the larger, is just visible to the naked eye and measures about $\frac{1}{70}$ of an inch (0.036 cm.) in length. It burrows in the corneous layer of the skin, deposits its eggs there, and dies in the burrow. The larvæ hatch from the ova in 5 or 6 days, become fully developed in 12 to 14 days more and then emerge to the surface of the skin. The male acarus is much smaller and does not burrow. The irritation caused by the burrowing results in the production of inflammation shown by papules, vesicles and pustules.

Symptoms.—The characteristic symptom is the presence of irregular elevated lines on the skin; the burrows made by the female parasite. These vary in size, the largest being about ½ inch (1.3 cm.) in length, and exhibit minute blackish or brownish spots, which are the eggs and excreta of the mite. At the end of the burrow there is a small elevated whitish spot beneath the skin; the female insect. The burrows are, however, often much outnumbered by the secondary multiform inflammatory lesions, namely the papules, pustules, vesicles, crusts, and excoriations. Pustulation is especially common in children.

The lesions of scabies always predominate in or are limited to certain regions of the body. These are the lateral surfaces of the fingers, the wrists, about the elbows, the axillæ, the lower part of the abdomen, the inside of the thighs, the ankles, and the feet; in short in regions where the skin is warmest and most delicate. In female patients the nipples are a favorite situation and in males the penis. The face always escapes, except in nursing infants infected from the mother. The lesions vary in number. In persons of cleanly habits who bathe daily there may be very few; in the dirty they may be numerous and widespread. Especially in these latter cases the burrows may be hard to find, on account of the number of inflammatory lesions present.

The itching accompanying scabies is very intense. This is particularly true in the night time, when, under the influence of the heat of the bed, the young acari migrate from the burrows. In persons with delicate skin eczema frequently develops as a complication, the result of the irritation by the parasites and the scratching. Urticaria, too, may occur.

Course and Prognosis.—The course is chronic, modified by care for cleanliness. There is no tendency to spontaneous recovery, but the prognosis is excellent under sufficiently thorough treatment.

Diagnosis.—This is often easy; sometimes difficult. It rests upon the frequent occurrence of several cases in one family; the severe itching developing some time after the patient becomes warm in bed; the distribution of the multiform lesions, particularly upon the flexor surfaces and elsewhere where the skin is thinnest, and upon the discovery of the burrows. These latter should be sought for especially between the fingers, on the flexor surface of the wrists and on the penis. *Pediculosis corporis* is distinguished, among other symptoms, by the fact that the hands are not attacked and by the discovery of pediculi in the underclothing. *Eczema* exhibits itching by day as well as by night; there is no special favorite situation, and there are present the infiltration and oozing characteristic of this disease.

Treatment.—Treatment is simple and effective if properly carried out. The patient should be thoroughly scrubbed with soap and water and given a hot bath. The parasiticide is then well rubbed in over the entire body except the head; and in cases occurring in infancy in which the face is involved, this part also must receive treatment. The anointing is repeated once or twice a day for 3 or 4 days, the frequency depending upon the development of irritation of the skin, there being no change made meanwhile in the bed-clothing or body-clothing. Following the last rubbing a hot bath is given and a complete change of bed-clothing and under-clothing made. After the lapse of 2 or 3 days an examination is made for any remaining evidences of the disease. Should these be discovered, a second course of treatment must be administered. The discarded clothing should be destroyed or very thoroughly disinfected. Attention should be given to the outer clothing. Of importance, too, is the careful search for itch in other members of the family, and the treatment of these. If this is not done, recurrence is almost sure to follow. As a parasiticide, one of the most popular is an ointment of precipitated sulphur. This may be given in the strength of 1 : 8. Another useful remedy is balsam of Peru, which may well be combined with the sulphur ointment (1 : 8), or used alone. Naphthol ointment (1 : 8) is also serviceable.

The treatment of scabies is very likely to produce a moderate amount of eczematous irritation of the skin, which will disappear readily under appropriate, mild treatment. It is important not to mistake this fictitious eruption for that of scabies, and to subject the patient to unnecessary second or more courses of treatment for the latter, inasmuch as this would only increase the irritation.

PEDICULOSIS

Etiology.—This extremely common affection is seen oftenest in those of any age where there is neglect of cleanliness. It is naturally more frequent in early life, because of the closer contact into which children come with others at school or in play. It is this ready communicability which is the frequent cause of the feeling of horrified surprise mothers of the upper class experience on the discovery of the parasites in the heads of their own children, whom they know they have kept clean. The fact must be accepted that the children of no class of society can be considered necessarily exempt.

The parasite is of three species; *pediculus capitis*, by far the most common, which attacks the scalp; *pediculus corporis* or *vestimenti*, infecting especially the undergarments, and *pediculus pubis*, attacking chiefly the pubic hair, but seen also on the eyebrows, eyelashes, the stiff hairs upon the body, and very occasionally upon the scalp. This last species is rare in children, and will receive no further consideration here. Pediculi nourish themselves by the sucking of blood from the skin through the proboscis. The ova or "nits" are deposited in numbers upon and are attached firmly to the hair-shafts, or, in the case of the body-louse, are found in the clothing. The lice hatch within a week and are mature in less than 2 weeks more.

Symptoms.—The chief symptom of *pediculosis capitis* is the itching more or less constantly present, which causes the child to scratch frequently at his head. This is often most marked in the occipital region, and not infrequently there develop patches of eczema or pustular lesions there or elsewhere on the scalp. The latter may even occur on the face and neck. The lymphatic glands of the neck especially of the occiput often become enlarged secondarily. Examination of the hair reveals the presence of the ova attached to the hair by one end. They are pinhead sized, oval, whitish bodies, or straw-yellow if the shell is empty or the embryo dead. There may be one or several on a hair. Often, too, live pediculi may be seen upon the scalp, or may be removed by a fine-tooth comb. The ova are further characterized by the firmness with which they adhere; this distinguishing them from the fine scales of seborrhea, which are readily removable.

Pediculosis corporis is often attended by intense itching and scratching as in the case of head-lice. The itching is most severe during the night, probably due to the fact that undressing dislodges some of the lice from the clothing, which then remain upon and irritate the body-surface. The lesions are most abundant where the clothing is most tightly pressed against the skin; as for instance over the shoulders and back, around the neck and waist, and on the outside of the thighs. The lice, if not numerous, are found only on the underclothing; the ova being attached to this and the parasites leaving it only to feed upon the skin. If in quite small numbers, they will be found only in the seams of the clothing.

Treatment.—The treatment of *pediculosis capitis* is generally promptly efficacious. In some instances, especially in girls with long hair, close cutting of this is advantageous; but although the procedure certainly renders the treatment easier it is not often required. Many remedies have been advised. Of these one should be employed which will not only kill the lice but destroy the ova also. If there is not too much cutaneous inflammation, the hair and scalp may be soaked with kerosene, and the head then wrapped with a towel to prevent the oil extending to the face and neck, where it may cause irritation. The towel can be left in place for 12 hours, and the scalp and hair then thoroughly washed. Needless to say the applications should not be made in the kitchen or near a flame of any sort. Accidents have happened from carelessness in this respect. The treatment can be repeated in a day or two if there is reason to fear that the disease is not cured. A useful lotion, if there are few open lesions, is one of glacial acetic acid minims 20 (1.23), bichloride of mercury grain ½ (0.032) and water fl. oz. 1 (30). This will even more surely destroy both the lice and the ova, the acetic acid having a softening effect upon the latter. The application should be preceded by a washing of the scalp and hair with soap and water, and the treatment should be given daily for a week. An old-time remedy is tincture of cocculus Indicus, diluted with 2 or 3 parts of alcohol and used daily. When there is much inflammation of the scalp an ointment is to be preferred, as of ammoniated mercury (grains 20 to 40:oz. 1) (1.3 to 2.6:31).

The treatment of *pediculosis corporis* is generally easy. The body should be washed thoroughly with soap and water and then disinfected with a stavesacre ointment (unguentum staphisagriæ), or a weak wash of bichloride of mercury. Particular attention must now be given to a very thorough disinfection of the clothing. Even the outer clothing is sometimes infected and needs baking.

BITES AND STINGS OF OTHER INSECTS

A number of other insects than the pediculus may excite disturbance of the skin. Of chief importance in this connection are fleas, bedbugs, mosquitoes, black flies, and the stinging insects such as wasps and bees. The **flea** (*pulex irritans*) produces a small erythematous elevation with a minute central hemorrhagic point. The parasite is widely diffused, but more abundant in some countries, especially tropical ones. It is much less common in most parts of America than in Europe. Many persons are entirely insensitive to the bite of this insect; others very susceptible, and in these an eruption of urticaria may be produced by the presence of but very few fleas. *Treatment* consists in the employment of lotions, such as of thymol, which are distasteful to the insect, as well as a relief to the itching. The wearing of cheese-cloth bags of camphor or pyrethrum under the clothing also serves, as a rule, to drive the parasites away.

The **bedbug** (*cimex lectularius*) produces by its bite a lesion like a flea-bite, but more inflamed, with a greater purpuric tendency and of longer duration. The treatment is that for urticaria. The **mosquito** and **black fly,** and the gnat and similar insects are the cause of great discomfort in many regions. The bite produces erythematous spots or wheals which are a source of intense itching in many individuals, and which may persist for some days. In others the irritation is but transitory and slight. The exposed parts are those attacked. A central point shows that the condition is a bite, and serves to distinguish it from other itching lesions. *Treatment* consists in the employment of antipruritic lotions suitable for urticaria or eczema. Camphor in oil, carbolic acid in alcohol (2 to 4 per cent.) and water of ammonia are serviceable applications for this purpose, apart from the careful use of netting. Ointments to prevent the biting may be employed, containing tar, menthol, pennyroyal, citronella and the like.

The sting of the **wasp** or **bee** causes decided swelling, redness and pain, the lesion having oftenest an urticarial appearance. If the stings are numerous, the constitutional symptoms may be severe. The old-fashioned prompt application of mud is one of the best to relieve the pain. In place of this ammonia water or a solution of carbolic acid in alcohol (2 per cent.) may be employed. Among the sources of irritation of a similar nature, but less often encountered, are the sand flea; the grain mite; the harvest bug; the spider; the ticks, and the hairs of certain caterpillars or moths, especially the brown-tailed moth.

CHAPTER II

DISEASES OF THE EYE

This topic is of so special a nature that it can be no more than touched upon, with the purpose merely of aiding the practitioner in diseases of children in the recognition and care of the simplest of these disorders. Special treatises upon these subjects must be consulted for further information. Ophthalmia neonatorum has already been discussed (Vol. I, p. 296); and various affections of the eye and ear connected secondarily with other diseases have been referred to under these separate topics. I am indebted to the text-books of DeSchweinitz, Packard, Biggs, Preysing, Posey and Wright and others, and to journal literature for valuable information freely used.

BLEPHARITIS

This disease depends upon general poor health; the exudative diathesis; pediculosis of the eyelashes; errors of refraction or eye-strain from other causes; the occurrence of diseases, such as measles, which are attended by inflammation of the eyes; eczema; and seborrhea. The **symptoms** consist in redness, swelling and scaliness of the margins of the eyelids, which in a more advanced state may develop into the formation of small crusted ulcers. The cilia are matted together; and on waking in the morning the lids may be adherent to each other. There is often considerable itching. In bad cases, if neglected, there may be permanent loss of the eyelashes through ulceration of the lids. Conjunctivitis is liable to be a complication. The course is often very chronic.

Treatment consists in removing the cause, as by improving the general health and by the fitting with glasses if any error of refraction exists. Conjunctivitis, if present, must receive appropriate treatment. Adhesion of the eyelids during the night may be prevented by rubbing the edges with petrolatum. The instilling every day or two of a 25 per cent. solution of argyrol is of value, with the frequent douching of the conjunctiva with a boric acid solution, or one of borax grains 6 (0.39); aq. rosæ fl.oz. 1 (30). One of the best applications to the lids is an ointment of yellow oxide of mercury and petrolatum (gr. 1 (0.065): dr. 1 (3.9). Any pustules which appear should be opened. In some cases thorough treatment for seborrhea of the scalp or face is necessary. If eczema is the cause, treatment appropriate for this must be employed.

HORDEOLUM

(Stye)

This is a localized inflammation of the edge of the eyelid, the starting point for which is the connective tissue, one of the follicles, or a sebaceous gland. It is often associated with blepharitis, and may be a further development of this, and its causes are the same. The active agent is generally a staphylococcus. The lesion is a hard, tender and painful swelling, finally with a yellowish point indicating suppuration. Some children have a remarkable tendency to suffer from this disease, while others are never affected. In the way of treatment hot fomentations are of service, followed by the incision of the stye when sufficiently softened. The yellow oxide of mercury ointment should be used as in blepharitis.

CONJUNCTIVITIS

Etiology.—Among the various causes of this affection, of which there are a number of varieties, are exposure to wind and dust; misplaced eyelashes; foreign bodies or other traumata; eyestrain; nasal catarrh; cutaneous or other disorders affecting the eyelids; the occurrence of various acute infectious diseases, especially grippe and measles; and impairment of the general health as in tuberculous or lymphatic subjects. Bacteria of various sorts are always found on the conjunctiva even in health. In some cases their presence would appear to be accidental, or at least in no way pathogenic. In others they are doubtless the specific cause of the inflammation. The relationship of the gonococcus to conjunctivitis has already been discussed. (See Ophthalmia Neonatorum, Vol. I, p. 297.) Among other specific germs are especially to be mentioned the Koch-Weeks bacillus seen in the so-called "pink-eye;" the influenza bacillus; the pneumococcus; the Morax-Axenfeld bacillus, and species of staphylococcus and streptococcus. There is a distinct contagiousness observed in some forms, and this is often very decided, causing epidemics of the inflammation to occur.

Symptoms.—The different varieties of conjunctivitis can be more conveniently considered separately.

Catarrhal Conjunctivitis.—This is the simplest form of the disease and occurs independently of the action of any specific microörganism. It is commonest in children and young persons. Its symptoms consist in injection of the conjunctival vessels of one or both eyes, both of the lids and of the eyeballs; more or less photophobia, itching or other discomfort about the eyes; and an increase of secretion. This secretion may be serous or mucopurulent, free or scanty. There may be slight haziness of vision. This disease is not contagious and the inflammation generally disappears in a few days.

Acute Contagious Conjunctivitis.—"Pink-eye," as this variety is often called, is a severer form of the malady just described; very contagious; occurring often epidemically at any age and oftenest in autumn or spring, and dependent upon the action of the Koch-Weeks bacillus. The symptoms, which begin in about 36 hours after exposure, consist of hyperemia and secretion, soon advancing to mucopurulent discharge; great injection of the vessels; swelling of the lids, and sometimes chemosis. There is a burning sensation of the eyes and the lids adhere when waking in the morning. Both eyes are attacked simultaneously, or one soon after the other. The duration is from 4 to 10 days, and the prognosis is good, as a rule, but careful treatment is often required.

Among forms of acute conjunctivitis dependent upon other causes are *influenzal conjunctivitis*, seen most frequently in infants and children, and pneumococcal conjunctivitis, occurring at any age, but chiefly in early life. The latter is to an extent contagious.

Angular Conjunctivitis.—A more chronic form, of insidious onset and less common in children, is angular conjunctivitis depending upon the Morax-Axenfeld diplobacillus. There is aching and a sensation of sand in the eyes, with redness of the edges of the lids, especially at the canthi, and congestion of the conjunctiva in the vicinity. The secretion is soapy, greyish, scanty, not purulent, and adheres to the edges of the lids, or is seen at the internal canthus. The lids stick together over night. The disease is thus a combination of a blepharitis and a limited conjunctivitis. In other cases the conjunctival inflammation predominates. The course is tedious, and the disease may last for months, even under treatment. Phlyctenules and corneal ulceration may occur as complications.

Vernal Conjunctivitis.—This form of the disease is characterized by the tendency to develop each year with the oncoming of warm weather. There is photophobia, lachrymation, and mucous secretion and discomfort in both eyes, with injection and thickening of the conjunctiva, and the formation of grey, flattened nodules on both the palpebral and ocular surfaces, but in the latter position only at the margin of the cornea and overlapping it. The condition is most common in childhood, and usually yields readily to treatment, but returns the succeeding year, and so on during several years.

Purulent Conjunctivitis.—This may be the result of either the action of the gonococcus or of other germs, especially the pneumococcus. The disease may occur at any age, and the symptoms, prognosis and treatment are similar to those discussed under ophthalmia neonatorum (Vol. I, p. 296).

Pseudomembranous Conjunctivitis.—This disorder may be either diphtheritic in nature, or depend upon a variety of other germs, and sometimes occurs in connection with measles, grippe and scarlet fever. It is commonest in early childhood. The diphtheritic form is contagious.

The *non-diphtheritic* type begins with the symptoms of acute conjunctivitis of one or both eyes, with a thin, seropurulent discharge, swelling of the lids, and in a few days a deposit of false membrane on the inner surface of the lids but not upon the eyeball, unless the streptococcus be the germ present. The prognosis is good under careful treatment. The disease lasts 2 to 4 weeks, providing the causative germ is other than the streptococcus. With this as the active agent, the prognosis is unfavorable; the inflammation being more severe, the lids more swollen and the discharge more abundant and thicker. The cornea may be destroyed, and even life may be lost.

Diphtheritic conjunctivitis produces a thin, scanty, sero-purulent discharge, thickening and rigidity of and pain in the lids, and false membrane upon the conjunctiva even of the eyeball. Ulceration of the cornea develops, and in bad cases sloughing. The constitutional symptoms are severe, and the prognosis is bad. The disease may be primary on the conjunctiva, but is oftener associated with nasopharyngeal diphtheria. Clinically there is practically no difference between this diphtheritic and the severer non-diphtheritic forms of pseudomembranous conjunctivitis.

Treatment of Conjunctivitis.—The treatment of *catarrhal conjunctivitis* consists, first, in removing the cause, and, second, in allaying the inflammation. The latter may readily be accomplished by frequent bathing with hot or cold water, guarding the eye from light with an eye-shade or with dark glasses, and the frequent douching, using an eye-cup, with normal salt solution, a solution of boric acid gr. 10 (0.65) : fl.oz. 1 (30), or one of borax (sodium biborate gr. 6 (0.39), aq. rosæ and aq. camphoræ each fl.oz. ½ (14.8)). Instilling daily a few drops of a 25 per cent. solution of argyrol is of service in cases at all obstinate. In some instances, too, benefit may come from the daily use of stronger astringent solutions, such as zinc sulphate (gr. 2 (0.13) : fl.oz. 1 (30)). The edges of the lids may be smeared with petrolatum.

Acute contagious conjunctivitis demands similar treatment, the argyrol or zinc sulphate solution being effective. Severe cases may have the lining of the lids lightly brushed with a 0.5 per cent. solution of silver nitrate, and the eyes irrigated several times a day with a 1 : 10,000 solution of bichloride of mercury. For *angular conjunctivitis* the best treatment is the solution of sulphate of zinc mentioned.

Vernal conjunctivitis requires the use of dark glasses, boric acid eyewash, and yellow oxide of mercury ointment. Owing to the obstinate tendency to recur, the case should be referred to a specialist upon diseases of the eye. In view of the serious character of *pseudomembranous conjunctivitis*, an ophthalmic surgeon should certainly be consulted if possible. The line of treatment consists in the employment of ice-compresses, keeping the eyes as clean as possible with boric acid solution or with one of bichloride of mercury (1:10,000), and the employment of argyrol (25 per cent) or of nitrate of silver 0.5 per cent. Diphtheria-antitoxin should be given promptly in all cases suspected of being diphtheritic. Atropine should be instilled, and the sound eye guarded by an occluding bandage.

PHLYCTENULAR CONJUNCTIVITIS AND KERATITIS

Etiology.—The causes of this affection are commonly constitutional. It is sometimes seen in subjects of the exudative diathesis, and it occurs in combination with tuberculous adenitis, adenoids, rhinitis, malnutrition, chronic digestive disturbances, or imperfect hygiene; or it may develop as a sequel to infectious fevers, especially measles. There is often an intimate association with eczema, and the malady has been called "eczematous conjunctivitis;" while by others it has been claimed to be a manifestation of the presence of the toxin of tuberculosis in the body, or even to consist of actual tuberculous foci.

Symptoms.—The phlyctenules consist of an infiltration of round cells, producing minute, grey, translucent elevations surrounded by injected vessels. In phlyctenular conjunctivitis these are situated chiefly on the bulb of the eye, near or on the corneal margin, or occasionally on the tarsal conjunctiva. In phlyctenular keratitis they are on the cornea itself. The nodules may be one or several. As the disease progresses, the apex of the phlyctenule softens and grows yellow, and an ulcer forms, which leaves no scar in the conjunctival cases; but which on the cornea often destroys the outer layer, and after healing may leave a small opacity. There is more or less lachrymation and pain, and photophobia is a very prominent symptom, especially in keratitis. In these severer cases the child may be entirely unable to open the eye on this account, and only forcible separating of the lids reveals the lesion.

The **prognosis** of the conjunctival cases is always good, and recovery occurs in from 1 to 2 weeks. In keratitis the prognosis is more uncertain and the course more varied. In the large majority the ulcers heal with no trace or with an almost undiscoverable scar. In more severe cases, however, the scarring may be decided and interfere with vision; and in some instances perforation of the cornea may occur and cause loss of sight. Cases of keratitis in which the so-called "scrofulous" symptoms are well marked (p. 500) often are long-continued and very resistant to treatment.

Treatment.—In the milder cases in which the lesions are confined to the conjunctivæ this consists in the frequent employment of soothing eye-lotions, as in other forms of conjunctival inflammation, and in guarding the eye from light by wearing colored glasses. Ointment of yellow oxide of mercury (gr. 1 (0.065) : dr. 1 (3.9)) should be introduced into the eye once a day after the acute symptoms have moderated; or calomel may be dusted over the lesions. When the cornea is involved one would do more wisely by entrusting the case to an ophthalmic surgeon if possible, as the final results are more uncertain and the difficulty in examining and making applications to the eye is often very great. Instillation of a solution of cocaine may relieve the pain and the resulting blepharospasm and permit of inspection and treatment. The eye should be douched several times a day with a boric acid solution and the application of one of argyrol is of benefit. The yellow oxide of mercury should be employed as in the conjunctival cases, or calomel be dusted over the cornea once daily, if the phlyctenules have ulcerated. Atropine should be instilled in sufficient strength and frequency to keep the pupil dilated. The external commissure is often fissured, increasing the blepharospasm; and touching this with sulphate of copper or nitrate of silver may relieve this trouble. Any postnasal growth or rhinitis present should receive appropriate treatment. In addition to these local measures it is imperative that every means be taken to improve the general health. Change of climate to the mountains or to the sea, provided all glare be avoided, is often one of the best measures when possible; and supervision should be employed over the hygiene and diet.

CHAPTER III

DISEASES OF THE EAR

OTITIS

Inflammation of the ear in some of its forms is of extreme frequency in early life, associated with many conditions. Reference has repeatedly been made to it in discussing special topics. It may be divided into inflammation, respectively, of (1) the external ear, (2) the middle ear, and (3) the internal ear.

1. Otitis Externa. **Etiology.**—The disease is of three forms, and the causes vary somewhat accordingly. (*a*) *Diffuse inflammation* depends upon an infection by pyogenic germs, sometimes arising spontaneously, sometimes following a trauma such as may be produced by foreign bodies and especially by the effort at their removal. The presence of insects in the ear is an occasional cause, as is the development of aspergillus here. Eczema is also an etiological factor, and the germs of Vincent's angina may attack the ear in debilitated children. (*b*) *Pseudomembranous otitis* is generally the result of the action of the diphtheria bacillus in the external auditory canal. (*c*) In a third form, the *acute circumscribed inflammation*, or *furuncle*, pyogenic germs are the cause as in furunculosis elsewhere in the body.

Symptoms.—In the *diffuse form* there is pain, itching, swelling and narrowing of the external canal. The latter results in some degree of temporary loss of hearing. There may be a thin or viscid discharge, which occasionally becomes purulent in chronic cases, and there is a thickening and a very free desquamation of the epithelium of the canal. Sometimes moderate fever is present. *Pseudomembranous otitis externa* is a rare disease, producing severe and often dangerous symptoms. There is swelling of the auricle, and narrowing of the canal by the formation of a pseudomembranous exudate; and there appears a thin, or later a purulent, discharge from the meatus, which causes excoriation of the skin of the neighboring regions followed by a pseudomembranous deposit there. The constitutional symptoms are severe.

The third form, the *furuncle*, is not very common in children. It produces pain, swelling, great tenderness in the canal, and sometimes temporary impairment of hearing. Finally, after several days of illness, pus forms and is discharged, unless earlier relieved by treatment. Other boils are likely to follow, thus prolonging the attack.

The **prognosis** in otitis externa is generally good, except for the danger of extension to the middle ear or the bony structures. The **diagnosis** is not always easy, on account of the difficulty attending the employment of a speculum. Generally distinct evidences are discoverable that the situation is external to the middle ear.

Treatment.—The cause is to be sought for and removed if possible. The next indication in diffuse inflammation is the controlling of the condition by the employment of moist heat, as by douching with a hot boric acid solution. In the pseudomembranous form it is safe to presume that diphtheria is the cause, and antitoxin should be given. Locally warm antiseptic applications should be employed, such as weak solutions of corrosive sublimate, carbolic acid or peroxide of hydrogen. For furunculosis an ointment of ichthyol may be tried; but generally the quickest mode of relief is incision when pus has been found. As a rule constitutional treatment is indicated, as in furunculosis elsewhere. (See p. 567.)

2. Otitis Media.—This extremely common affection of infants and children is generally divided into the *catarrhal* and the *purulent* forms. While a large number of catarrhal cases terminate in suppuration, the purulent form is not always preceded by a catarrhal stage. In catarrhal otitis the process is similar to the catarrhal inflammation seen in the mucous membrane of the nose and elsewhere and is nonsuppurative. We may conveniently study the two subdivisions together.

Etiology.—Age is a prominent predisposing factor, the large majority of cases occurring in infancy and childhood. The cold season of the year predisposes also. The inflammation is nearly always secondary to disease elsewhere. Very often it is an extension by way of the Eustachian tube from a catarrhal inflammation of the nasopharynx. This occurrence is rendered particularly easy in early life by the short length and relatively greater diameter of the tube. It is especially common if adenoids are present. It may be produced also by the nasal douche or syringe forcing secretion through the Eustachian tube into the middle ear. Pneumonia, chiefly bronchopneumonia, is often attended by otitis; and in marantic infants with tuberculosis, syphilis, or gastrointestinal disturbance its occurrence is of very great frequency. Various acute infectious diseases predispose to inflammation of the middle ear, prominent among them being scarlet fever and measles, although diphtheria, grippe, typhoid fever, variola, varicella, pertussis and cerebrospinal fever are common causes. The frequency of otitis in these different diseases has already been referred to to some extent in discussing the individual disorders. In a large series of nurslings Scherer and Kutvirt[1] found otitis media in 18.6 per cent. of those with disturbances of digestion; 12.2 per cent. of the pharyngeal cases; 6.66 per cent. of those with pneumonia; and 22.5 per cent. of the congenitally syphilitic.

The direct cause in the purulent cases is the action of bacteria of various sorts, prominent among them being the pneumococcus, streptococcus, staphylococcus and influenza bacillus, alone or in combination. The most severe form is caused by the streptococcus. The tubercle bacillus is sometimes found in the chronic suppurative cases. Much the most frequent germ observed, especially in infancy, is the pneumococcus. In 154 examinations Preysing[1] discovered the pneumococcus 112 times, being in pure culture in 96 instances. The germs enter the ear through the Eustachian tube.

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[1] Jahrb. f. Kinderh., 1915, LXXXII, 249.

Pathological Anatomy.—The first lesions occurring are swelling and injection of the mucous membrane lining the tympanic cavity, the process having extended from the nasopharynx by way of the Eustachian tube and having partially or completely closed this. In the catarrhal cases more or less serous or mucous exudation takes place. Those which advance beyond this point are purulent, the fluid containing pus-cells, red blood-corpuscles and epithelial cells. In the severer cases the inflammation is of a more virulent type; numerous bacteria are present; the tympanic cavity is filled and the membrane perforates; and the inflammation may extend to the bony tissue and even thence to the meninges and the brain-substance. In cases of chronic catarrhal otitis, there is an exudative inflammation followed by connective-tissue changes with adhesions, or the condition may develop without initial effusion.

Symptoms.—The disease, as stated, is generally divided into the *catarrhal* and the *purulent* forms, either of which may be acute or chronic; yet no sharp distinction can be made unless perforation of the drum-membrane occurs and the presence of pus becomes evident. Either one or both ears may be involved, although not necessarily to the same degree or simultaneously, and the attack may be over in one ear before the other is affected. Bilateral otitis is especially common after the acute infectious diseases. In the 217 cases of otitis in infancy reported by Scherer and Kutvirt[2] 100 were bilateral. With remarkable frequency the diagnosis of otitis is not made until perforation occurs, the symptoms being masked by those of the primary disease, or being supposed to depend upon other causes. On this account it repeatedly happens in pneumonia, typhoid fever, scarlet fever, diphtheria and measles, that an otitis may be entirely overlooked until an examination of the ears is made, or until rupture takes place. In typical acute cases in which this masking does not occur, one of the most characteristic symptoms is the development of fever, which, although usually moderate, may reach 103° or 104°F. (39.4° or 40°C.), usually with decided fluctuations in its course. With the fever there is evidently pain located in the ear, usually only on one side, as indicated by children who can speak, and sometimes equally well by infants through the putting of the hand to the side of the head, and the very evident tenderness shown when the ear is pressed upon. Sometimes the pain is almost continuous; at other times more intermittent, the child waking from sleep with a start and crying. The pain is quite commonly worse at night, and the patient may be reasonably comfortable during the day-time. Nothing definite is discovered on general physical examination of the body, although the infant seems decidedly ill; but should an examination of the ear be made, there will be found decided redness of the tympanic membrane, and later a yellow coloration with distinct bulging. Some impairment of hearing is a very common early occurrence, liable to persist for some days after the acute symptoms are over. Examination of the blood usually reveals a leucocytosis.

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[1] Otitis media der Säuglinge, 1904, 5.

[2] *Loc. cit.*, 138.

The symptoms as described may be considered representative of the typical condition, but there are so many variations from it seen so frequently, that the diagnosis is often obscured. In atrophic infants suffering from gastrointestinal disturbances there may be no symptoms whatever discoverable connected with the ear, and the attendants are surprised some morning by finding pus in the auditory canal and upon the pillow. In other cases there is decided fever, but no evidence of pain or tenderness about the ear, and consequently no noteworthy crying, although the children are restless and sleep badly. In fact the only suspicious symptom is the continued fever without discoverable cause. In still other instances the persistence of fever with no pain located in the ear is supposed to depend upon a prolongation of the primary disease, and a correct diagnosis can be made only by an aural examination; while in others there are symptoms strongly suggesting meningeal irritation, such as vomiting, irregular pulse, hebitude, delirium or convulsions.

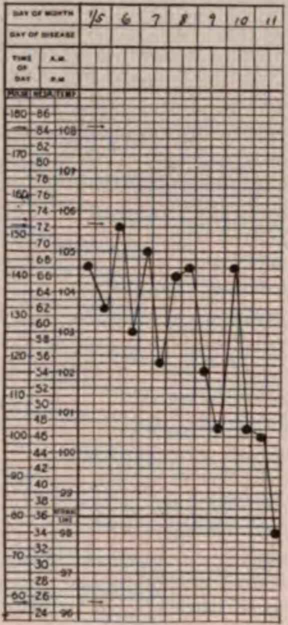

Fig. 434.

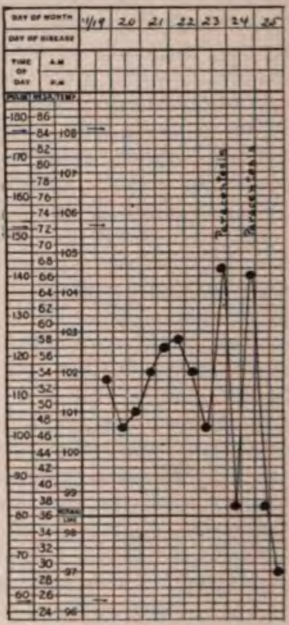

Fig. 435.

Fig. 434.—Otitis Media.

Gertrude P., aged 11 months. Admitted to the Children's Hospital of Philadelphia, Jan. 2, with physical signs and symptoms of pneumonia; Jan. 8, otitis discovered, paracentesis performed, with prompt fall of temperature; Jan. 10, rise of temperature followed by fall with the development of free discharge from the ear.

Fig. 435. Otitis Media.

Mike P., aged 15 months. A poorly nourished infant, ill 1 week with cough, fever and diarrhea. Admitted to the Children's Hospital of Philadelphia, Nov. 19. Congestion of the drum-membrane discovered, leucocytes 27,400; Nov. 23, bulging of drum-membrane, paracentesis done, with prompt fall of temperature; Nov. 24, rise of temperature, paracentesis again performed, followed by disappearance of fever.

Probably in the majority of recognized cases, and certainly if one includes all those which have been unrecognized, the disease does not go on to suppuration and perforation, but resolution and absorption of the fluid take place. This often occurs after 1 or 2 days, there being no pain or fever after the first night. Often the course is more prolonged, exacerbations occurring every night, or perhaps with a day or two of a free interval; and after all the acute symptoms have disappeared the presence of fluid in the tympanic cavity may be discovered on otoscopic examination.

In the more severe acute cases which advance to suppuration the rapidity of the process shows a great variation. Not infrequently perforation and discharge of pus may occur within a few hours from the onset of symptoms. In other instances this may not take place for some days or even weeks after the acuteness of the symptoms has subsided to a considerable degree. The fever in the suppurative cases is often higher and shows a more decided tendency to irregularity; the pain is often more severe; there is frequently tenderness over the mastoid process as well as in front of the tragus, and the glands in the neck may be enlarged. After the discharge of the pus the temperature generally falls rapidly and pain disappears (Figs. 434; 435). It is, however, by no means always true that fever ceases; and even without evidences of complications an irregular elevation of temperature may sometimes continue for days after perforation has taken place, and be a source of anxiety. Deafness, too, which is often quite decided before perforation occurs, does not disappear immediately.

The majority of perforating cases do not go beyond the acute stage, but in others the disease passes into a *chronic purulent otitis media*. This is especially common in patients with tuberculosis evident or concealed in the system, as also after attacks of measles, diphtheria, and scarlet fever. In tuberculous subjects purulent discharge from the ear may continue indefinitely, perhaps with short or long intermissions, the perforation in the drum-membrane remaining open. There is no pain or fever, but complications may readily develop. The discharge from the ear often has a very offensive odor.

Chronic catarrhal otitis media is generally bilateral. It may follow repeated attacks of acute catarrhal otitis, or may arise insidiously as a sequel to chronic diseased conditions of the nasopharynx, including adenoid hypertrophy. The most prominent symptoms are deafness and ringing in the ears. There is generally no pain. The Eustachian tube is not patulous, or only partially so, and the tympanic membrane is usually grey, apparently thickened, and retracted. The disease is less common in children than later. The deafness generally progressively increases, and if the process has begun sufficiently early in life, deaf-mutism may result. Occasionally a chronic purulent otitis is followed by a chronic catarrhal condition.

Complications and Sequels of Otitis Media.—These are so likely to occur and so important that the possibility of their development must not be forgotten, and a careful search must be made in every instance. The most important of them are the following:

1. *Mastoiditis.*—This is the most common of the inflammatory complications. It occurs acutely with especial readiness in early life, on account of the ease with which pus enters the mastoid antrum at this age, the mastoid process containing one large cell connected directly with the tympanic cavity instead of the numerous small, closed cells

which are present in adult life. It is most often seen in the 2d year. A mild type is probably a frequent attendant upon purulent otitis at any time of life, as shown by otoscopic examination or by the tenderness often present over the mastoid process. It does not, however, in this event produce special symptoms, inasmuch as the pus is discharged through the auditory canal.

The disease in its well-marked form is made more frequent than need be by the neglect of effective treatment in cases of purulent otitis. The *symptoms* consist of fever, pain and tenderness in the mastoid region; and later edema and swelling of the skin here, with a pushing of the auricle away from the head (Fig. 436). Its existence is to be suspected whenever the temperature continues elevated after the drum has been opened, or when the flow of pus from the auditory canal ceases but other symptoms of otitis persist. It may develop while the otitis is still active, or oftener 2 to 3 weeks after discharge from the ear commenced. In many cases there is but little fever, and in infancy the determination of the presence of local pain is often very difficult. Examination of the ear shows an unusual shortness of the canal, a depression downward and forward of its postero-superior aspect, and a bulging or a discharging perforation of the tympanic membrane.

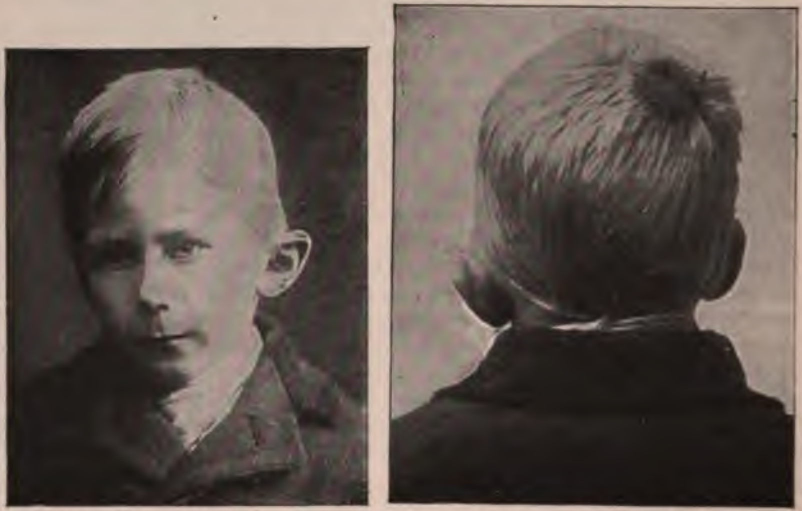

FIG. 436.—FRONT AND REAR VIEWS OF A CASE OF ACUTE MASTOID PERIOSTITIS.

(*Alderton, in Posey and Wright, Treatise on Diseases of the Eye, Nose, Throat and Ear* 1903, 1146.)

2. *Thrombosis of the Lateral Sinus.*—This may occur as a sequel to mastoid disease, but is uncommon. It may develop after operation upon the mastoid process, or independently of this. The symptoms have already been described in discussing Sinus Thrombosis (p. 342).

3. *Meningitis.*—A serous meningitis may occur, due to the neighboring presence of pus; or the pus may actually make its way through the wall of the mastoid process, or from the roof of the tympanic cavity, to the meninges, and produce a localized or more general septic meningitis. In other instances meningitis results from the rupture of an otitic cerebral

abscess, or follows the development of a septic thrombosis of the lateral sinus. It is not a frequent complication especially in infancy. The symptoms have already been described (p. 324).

4. *Abscess of the Brain.*—This rare complication is produced in the same way as is septic meningitis, with which, or with sinus thrombosis, it may be combined. It is found oftenest in the temperosphenoidal lobe or in the cerebellum (see Abscess of the Brain, p. 348), in the former region when the disease began in the middle ear (Körner);[1] in the latter when there is chronic suppuration in the labyrinth. The *labyrinth* is only exceptionally involved as a complication to otitis media.

5. *Facial Paralysis.*—This is a complication not infrequently observed, oftenest developing in the course of the purulent form of otitis media. It is produced by involvement of the facial nerve in its canal, as a result of extension of the inflammatory process from the bone. (See Facial Neuritis, p. 402.)

Prognosis.—This depends to a certain degree upon the cause of the disease, and largely, too, upon the variety of inflammation present. In *acute catarrhal otitis* the prognosis is nearly always good under careful treatment both of the condition itself and of its causes. The acute symptoms cease in 1 or 2 days, and any fluid present is rapidly absorbed. There is always danger, however, without such treatment that some degree of permanent deafness may remain. This is especially true if repeated attacks occur. *Acute purulent otitis* gives a prognosis usually excellent as regards final complete recovery, but to this there are many exceptions. Generally the discharge of pus continues for 2 or 3 weeks or less, the rupture in the tympanic membrane heals rapidly, and hearing is completely restored. However, after the acute infectious fevers, especially measles, diphtheria and scarlet fever, or in tuberculous subjects, there is always great danger of the process becoming chronic. In *chronic catarrhal otitis* resulting from earlier acute catarrhal attacks, or from chronic catarrhal disorders of the nasopharynx, the prognosis is uncertain and often unfavorable as far as hearing is concerned. It depends not only upon the local condition, but upon the general state of health. In the chronic catarrhal condition which sometimes follows a chronic purulent inflammation connective tissue adhesions may remain and permanent deafness result. The prognosis of *chronic purulent otitis* is uncertain. Often the discharge continues perhaps for years in spite of treatment; and even after its cessation the perforation does not close. The structures of the middle ear may have been seriously damaged, and deafness may be permanent, but can generally be improved under treatment. As long as the suppurating process persists either in the acute or the chronic purulent cases there is danger of the development at any time of mastoiditis, meningitis, and similar processes.

It is impossible to predicate in any case of otitis whether mastoiditis will develop as a complication; and in just what percentage of cases it will occur is uncertain. It is beyond question that it is not infrequent in a state readily recognized, and equally so that systematic examination of the ears would show its presence in a larger number.

Diagnosis.—The diagnosis is easy in many instances; while in numerous others it is not made until discharge from the ear occurs. There is little, as a rule, in the cases in atrophic infants to call the attention to the ears; and in otitis in cases already exhibiting fever the presence of this symptom cannot serve as a diagnostic guide. In infancy the occurrence of fever and restlessness, without recognized pain, and without sufficient discoverable cause, should always lead one to an examination of the ears. This is even more imperative if there is clearly pain in the ear. In the catarrhal cases the diagnosis rests upon the tenderness in front of the tragus, the pain, and the redness of the drum-membrane, especially in the peripheral zone and over the handle of the malleus. There may also be moderate bulging without fever in the later stages. In the suppurating cases the course is more severe, the fever higher and more irregular, the lymphatic glands of the neck are often swollen and the drum-membrane is dull, bulging and sometimes yellowish in color.

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[1] Lehrb. d. Ohren- Nasen- u. Kehlkopfkr., 1909, 341; 345.

Treatment.—Naturally this varies with the type of the otitis and with the case; and in all serious conditions it is much better to refer the patient to the care of an aural surgeon. *Prevention* is of great importance. In cases with any tendency to recurrent otitis, enlarged tonsils and adenoids should be removed if present; and any catarrhal condition of the nasal and pharyngeal mucous membrane should receive appropriate treatment. During the infectious fevers, especially diphtheria, scarlet fever and measles, the mouth should be kept clean and the nasal passages clear by gently spraying or douching with a warm normal saline or a mild antiseptic solution. The possibility of driving infectious material into the Eustachian tube and the middle ear is a real one; but probably the danger from not using local treatment is greater.

Usually the first indication in an attack of *acute otitis* is the relief of the pain. For this purpose may be employed external heat, as with the hot-water bag, or by the forcible injection into the auditory canal of water as hot as can be borne. A few drops of a mixture of cocaine and adrenaline is often useful instilled into the auditory canal, using 3 to 5 minims (0.185 to 0.31) of a 4 per cent. solution of cocaine in a 1 : 1000 or 1 :3000 solution of adrenaline chloride. In addition the nose should receive treatment for the acute rhinitis often present, and the instilling of a few drops of a weak mixture of cocaine and adrenalin, followed by spraying with an oily solution aids in contracting the nasal mucous membrane and giving access of air to the Eustachian tube, with the subsequent discharge of fluid from it. Caution must be observed, however, in the nasal application of cocaine in young children. Inflation of the ear-drum by the use of the Politzer bag, after previous cleansing of the nasal passages, is often of benefit after acute symptoms are over, and will aid in restoring the hearing. Internal measures should be taken to reduce the inflammation, as by the employment of aconite, belladonna, and purgatives. If the pain is not relieved promptly the administration of an opiate is necessary and should not be omitted.

Meantime, of course, an examination of the ear should be made. A very decided degree of bulging, combined with the existence of fever and other symptoms, demands paracentesis. This performed early limits the duration of the pain and inflammatory symptoms, and tends to prevent the occurrence of serious local or more general injury. If there is tenderness over the mastoid, paracentesis should be done immediately. The wound heals in a day or two in the milder catarrhal cases. In others pus continues to be discharged for some time. After paracentesis or spontaneous perforation the ear should be syringed gently with a warm 1 : 10,000 solution of bichloride of mercury, or a saturated solution of boric acid; and this may be done 2 or 3 times a day, should the case be a purulent one, using a small syringe of soft rubber. If there is much secretion with an offensive odor, the cleansing is aided by using peroxide of hydrogen. After this procedure, boric acid in powdered form may be inserted into the canal, or a wick of cotton or gauze placed in position, either dry or moistened with a 10 per cent. carbolized glycerine. When the acute symptoms are over and the perforation has healed, the employment of the Politzer bag, as already referred to, hastens the recovery from the temporary deafness which is liable to remain.

The management of *chronic catarrhal otitis* of the dry form is troublesome and slow in its results. It involves a careful thorough treatment of the conditions of the nasal passages which are productive of the aural symptoms; a constant effort to maintain or improve the general health; and the employment of local measures, which should be conducted by an aural surgeon.

Chronic purulent otitis also is a disease requiring so much care in the details of treatment that the reader is referred to text-books upon otology. As in the last-mentioned form of otitis media, an aural surgeon should take charge of the case, if possible. Regarding the treatment of *mastoiditis*, the most important consideration is that prompt and free drainage be given to the mastoid cells. This can often be accomplished by paracentesis through the drum-membrane; and when this is not satisfactory operation upon the mastoid may become necessary. To determine what cases should undergo this operation requires much judgment and experience and the advice of an aural surgeon should certainly be obtained. Without doubt under the use of leaching or ice-bags, many cases recover without operative interference.

3. Otitis Interna (*Inflammation of the Labyrinth*).—This not very common affection may occasionally result from extension of inflammation from the middle ear, causing more or less destruction of the labyrinth. In other cases it follows some acute infectious disease, especially cerebrospinal fever, scarlet fever and mumps. The acute symptoms are usually not recognized, being concealed by those of the primary disorder. The principle symptoms discovered later are vertigo and deafness, and frequently nystagmus also. The deafness is often an absolute one, depending upon an involvement of the nerves. There is an absence of bone-conduction. Occurring early in life and in both ears, it is one of the causes of deaf-mutism. (See p. 280.) The prognosis is unfavorable, and treatment usually of little avail.

FOREIGN BODIES IN THE EAR

The **nature** of foreign bodies discovered in the auditory canal is very varied. They may find entrance either accidentally, or may have been pushed into the ear by the patient. Insects of different sorts may make their way into the auditory canal, or their eggs may have been laid there and the larvæ subsequently develop. Peas, pieces of wood, buttons, and the like, may have slipped from the patient's fingers, or moulds of different sorts may have grown in the canal.

If the object has not penetrated far, there may be no **symptoms** whatever, or only a moderate sense of tickling or discomfort, combined with impairment of hearing if the passage is entirely occluded. If deeper in the canal severe pain may be present, or inflammation may be produced be the presence of the body, and a liquid discharge take place. If the object has been pushed far in by unskillful efforts at its removal, the membrana tympani may be injured and inflammation of the middle ear may follow.

In the way of **treatment,** injections of warm, sterile water will generally be successful in removing the body if it is still in the membranous portion of the canal. The stream of water should be directed forward and downward, at the same time pulling the auricle upward and backward. An anesthetic should be given if necessary. If the canal is much swollen, and the object of considerable size, removal may be accomplished by employing delicate forceps, a blunt curette, or a hook. If this is not successful a more radical operation is required.

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